OCULAR PROBLEMS AND THEIR MANAGEMENT IN PAEDIATRIC PATIENTS OF TUBERCULOUS MENINGITIS IN BUNDELKHAND REGION

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C E R T I F I C A T E

Certified that the work entitled " OCULAR PROBLEMS AND THEIR MANAGEMENT IN PAEDIATRIC PATIENTS OF TUBERCULOUS MENINGITIS IN BUNDELKHAND REGION " which is being submitted as thesis for M.S.(Ophthalmology) examination of Bundelkhand University, 1985 by Dr. AJAY KUMAR SAXENA has been carried out in the Department of Ophthalmology.

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Dated 30.7.84

C E R T I F I C A T E

This is to certify that the work entitled " OCULAR PROBLEMS AND THEIR MANAGEMENT IN PAEDIATRIC PATIENTS OF TUBERCULOUS MENINGITIS IN BUNDELKHAND REGION " which is being submitted by Dr. AJAY KUMAR SAXENA , as a thesis for M.S.(Ophthalmology) examination of Bundelkhand University 1985, was carried out under my personal supervision and guidance. Examination of patients was done by candidate himself and the observations recorded has been checked by me time to time.

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A C K N O W L E D G E M E N T

I thank the Almighty for the successful completion of my thesis because God has been the source of inspiration from start to finish. Today, I again pray to the omnipotent God to give power to my pen sothat, I may be able to write a few words in praise of those persons, who have helped me in writing this manuscript from the very beginning right upto the end, though I can never replay the debt of their timely help in these words.

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At last but not the least, I thank to the little innocent babies, children and parents, without whose co-operation this study could not be made a success. I shall remain thankful for ever.

Dated: 30.7.84

(AJAY KUMAR SAXENA)

DEDICATED
TO
BABY RUCHI

IN HER
EVERLOVING MEMORY

(22nd NOVEMBER 1979 – 25th MAY 1984)

WHO

HAS BEEN SNATCHED FROM US
BY
CRUEL HANDS OF DESTINY

C O N T E N T S

INTRODUCTION

INTRODUCTION

Tuberculosis is known to have been one of the oldest of human diseases. It is as old as human hatred and hypocrisy. It has been appropiately described as " the captain of all the men of death " by John Bunyan (17th centuary). Few diseases have had such extensive or such intensive impact on the sober consciousness of mankind as tuberculosis. In no other disease has there been such a spectacular reversal of dread and danger as when, for the first time, truly effective remedies emerged on the chemotherapeutic horizon some four decades ago. In another couple of years there was a steep and striking decline in the attack rate. In countries which had the knowledge, the money and the men, tuberculosis was soon listed for ignominious retirement, though alas most of the poorer countries lagged, and still lag, tragically behind. India is one of them. The size and extent of the problems of tuberculosis in children seems to be directly linked to the prevalence of pulmonary tuberculosis among adults in community.

More than 200 years after the first description by SIR ROBERT WHYTT in 1768 tuberculous meningitis continues to be a serious problem. Of all the manifestations of tuberculosis, tuberculous meningitis is the most dreaded complication and a common cause of prolonged morbidity and mortality among children. Not with standing the availability

of potent and specific drugs, fatality rate of tuberculous meningitis still remains high as compared to other forms of tuberculosis in children.

With the advent of antitubercular treatment life, in a child of dreaded disease like tuberculous meningitis, may be spared, but innumerable crippling sequelae are seen. One of the important sequelae is in the form of BLINDNESS - total or partial leaving the patient as a walking and talking HEALTHY CADAVER.

Ocular changes form an important group of clinical manifestations of tuberculous meningitis. Moreover in those who servive, the disease might leave its permanent and dreadful hallmark in the form of partial or total blindness. Within the past years cases of these ocular manifestations have been reported in the medical literature, but these complications are still disastrous, as once the sight is lost the patient is handicapped in all activities. Keeping in mind all these facts, early detection of ocular complications and their management in tuberculous meningitis still required a review.

Tuberculous meningitis may produce a variety of changes in the form of intra or extra ocular manifestations having a sinister significance. These ocular changes may either manifest as papillitis, papilloedema or optic atrophy or there may be partial ocular pareses.

In the light of these dreaded ocular complications of tuberculous maningitis, the present study was undertaken to find out the prevalence of ocular manifestations of tuberculous meningitis in paediatric patients of Bundelkhand region, to make an early diagnosis and to prevent the dreading sequelae of BLINDNESS by early detection of ocular complications and their management.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY

Tuberculosis and tuberculous meningitis

Tuberculosis, one of the major public health problems in the developing countries of the world today has made its impact felt throughout the ages. The ancient writings of Indian medicine reveal that tuberculosus has been existing in India for more than 2000 years Park and Park (1976) have mentioned that a detailed description of tuberculosis has been found in apics written by past masters like Hippocrates, Charak and Susruta but the cause of this disease has been steeped in ignorance and superstition. The authors have also refferred the disease " Captain of the man of death" and " the great white plaque ". Authors further quote Hippocrates (400 BC) the father of medicine as calling it " phthis " which means " to dry up ". Robert Koch disclosed to the world on March 2, the tubercle bacillus.

Robert Whyth (1768) published the first clinical description of tuberculous meningitis in his monogram on " Dropsy in the brain " in children. Meindl (1982) described in his article that 150 years ago, the term TBM was not used. The term used most frequently for children was acute hydrocephalus. Two centuries age knowledge on the growth and development and health of children was quite rudimentary. In the 1803's the condition known as acute hydrocephalus and

chronic hydrocephalus were viewed as two forms of the tuberculous meningitis. The decisive motion to abandon the use of the term hydrocephalus was made in 1836 by Green. He introduced the term tuberculous meningitis (TBM). Ghai (1977) and Satya Gupta (1978) have commented that tuberculous meningitis is the most dreaded complication of tuberculosis and is the most common cause of death in infants and children suffering from tuberculosis.

Ocular manifestations

Duke Elder & Scott (Neuroophthalmology Vol.XII, 1971) have mentioned the work in the field of ocular manifestations of TBM, carried out by the workers in past. The work described in past is as follows -

The condition was produced experimentally by Deutschmann (1881), who injected pus from a tuberculous joint into the cranial cavity of rabbits and produced a clinical picture of papillitis and chorodal tubercles, necropsy showed a wide spread tuberculous meningitis, a perineuritis where in the dural and pial sheaths were studied with tubercles, and a spreading peripheral neuritis. Chiari (1877), Sattler (1878), Sisaric (1921) and Igerscheimer (1926) recorded an analogus process in clinical cases.

Saenger (1879) and Luce (1903) described involvement of cranial nerves in tuberculous meningitis Urbanek (1927) and Fronimopoulos (1938) reported that in tuberculous meningitis the sixth nerve is particularly liable to be involved and may be the first sign of the disease.

Incidence

The first national sample survey carried out by Indian Council of Medical Research (1958) showed the prevalence of tuberculosis to be about 7 per 1000 in children 5 to 14 years of age. of these 0.3 per 1000 were infectious. Subsequent surveys conducted by Pamara (1973) in Delhi and another one at the National tuberculosis institute, Banglore in the year 1974, have shown a significant and continuous decline in the disease in the children during the last 10 years or so.

Sheth (1961) reported that TBM constituted 4 to 8% of total admissions in pediatric wards. Udani (1961) reported that TBM was the second cause of admissions in preschool children and accounted for 22.1% of total admission in this age group Bharucha et al (1967) in a 4 year old study conducted at the K.E.M. Hospital, Bombay from 1961-64, reported the incidence of tuberculous meningitis to be 4 percent of all the paediatric admissions of a large teaching hospital. Bhakoo & Gupta (1969) reported the incidence of CNS tuberculosis as 1.8% from a study conducted at chandigarh. Rao (1972) reported that in different studies conducted in different parts of India, the incidence of TBM was 1-4% of the total in patient admissions.

FACTORS INFLUENCING MORTALITY AND MORBIDITY IN TERMS OF NEUROLOGICAL DAMAGE

In the Indian literature, Manchanda and Lal (1966) reported the maximum mortality (71.9%) and Kapur (1969) reported minimum mortality (17%) in children suffering from TBM. Between these two variable results have been reported by various other workers in the field.

Dikshit and Singh (1976) studied one hundred children to find out the various factors that influence mortality and morbidity in terms of neurological damage. It was observed that mortality and morbidity was highest if the child was less than 3 years of age, in IIIrd stage of disease (MRC classification), malnourished, belonged to low socioeconomic group, BCG test negative and suffered from pertussis or measles as a preceeding illness or presented as gastroenteritis. In their study poor nutrition cases have more neurological damaged 59% when compared with cases of fair nutrition 29% upto 3 years of age 75% cases had optic atrophy and 63% had mental retardation.

Gupta and Chopra (1981) commented that various factors influencing mortality and morbidity in terms of neurological damage are primarily dependent on the stage of the disease in which the treatment is started. Highest mortality as well as neurological damage occur in the IIIrd stage of the disease. The high mortality and neurologic damage in children of low socioeconomic status is probably

due to malnutrition, overcrowded living conditions, ignorance, illiteracy, poverty and failure to get prompt and adequate therapy. Antecedent infections like measles and pertussis have an adverse influence probably because of the depressed immunological status and rapid dissemination of infection.

PHYSICAL CHARACTERISTICS (CLINICAL FEATURES):

TBM is usually insidious in onset but may be fulminant if a caseous lesion discharges directly into the subarachnoid space. Heinz & Thomas (1979) grouped the clinical manifestations of TBM into 3 stages.

Stage I (General, nonspecific symptomatology).
Stage II (Appearence of definite neurologic signs), and
Stage III (Coma).

Stage I - This is characterised by irritability disinterest in play, idly staring in space, fever, headache, vomiting, anorexia and constipation.

Stage II - This is characterised by convulsions, signs of meningeal irritation, cranial nerve palsies and features of buldging anterior fontanelle or crack pot sound over head Fundus examination may reveal papilloedema and optic disc may show primary or secondary optic atrophy.

Stage III- In this stage there is unresponsive opisthotonous, The most common neurologic sequelae are developmental retardation, cranial nerve palsies, hydrocephalus, optic atrophy, deafness, paralysis, continuing stupor or coma, convulsions and pituitary disturbances.

CLINICO-PATHOLOGICAL ASPECT :

Rich & Mcordon (1933) suggested that after the initial bacillemia, granuloma formation (sometimes referred to as Rich foci) occurs in the meninges and for reasons that are unknown, these granulomas break down at a later date in susceptible hosts and tubercle bacilli spills into the subarachnoid space producing meningitis. Macgregor & Green (1937) reported that 78 patients out of 88 cases studied had granulomas in the brain, spinal cord, meninges or in combination of these. Tandon (1973) commented that meningeal exudates give rise to meningeal signs, cranial nerve palsies and hydrocephalles. Lesions in the brain parenchyma causes alteration of sensorium, seizures, hypothalmic symptoms and brain stem disturbances. Arteritis causes vascular obstruction and focal neurologic deficit. Allergy and hypersensitivity cause oedema of the brain.

Gupta & Chopra (1981) described that TBM is never a primary manifestation but always occurs as a result of secondary haematogenous dissemination from the site of primary extra cranial tuberculosis lesion which is frequently in the lung.It usually occurs with in the first 6-12 months after the primary infection. As a result of hematogenous dissemination the tubercle bacilli are lodged principally at leptomeninges and brain parenchyma.

Dastur et al (1983) studied 100 autopsied cases of TBM including 78 children. They have shown different mechanisms to produce brain damage. These pathogenic factors are the meningitis itself, the infiltration of the brain by meningeal exudate causing borderzone encephalitis, the involvement of large & small vessels by the meningeal exudate resulting ischaemia and infarction ; the internal hydrocephalus ensuing from the blockage of C.S.F. pathways, most commonly the basal cisterms by exudates; the presence of intraventricular exudate constituting epen-dymitis and subependymitis; the development of large or small brain tuberculomas and the occurence of brain oedema.

Pathogenesis of ocular involvement -

Boyd (1945) described that interstitial optic neuritis may result secondary to tuberculous meningitis leading to optic atrophy. Taylor et al (1955) commented that optic atrophy results from end arteritis obliterans involving the arteries supplying the optic nerve.

Mooney (1956) observed that the pathologic process in reversible oedema is external hydrocephalus whereas in progressive papilleoedema the tuberculous archnoiditis causes papilloedema by interfering with the circulation of C.S.F. The pale disc results from strangulation of pial vessels going to optic nerve & Chiasma.

Walsh (1957) described that widely dilated pupils become marrowed by lumbar puncture but return to original size soon after. It has been argued that release of pressure by lumbar puncture relieves the cerebral irritation and thus the pupils contract, but with refilling of the ventricles, the pressure rises again and pupils dilate.

Bhatnagar & Srivastava (1961) reported that paralysis of ocular muscles results from infiltration of III and VI nerve at the base of brain or it may result from raised intracranial tension. The aetiology of retrobulbar neuritis is by involvement of the region of optic Chiasma and progression of inflammatory process forward along the optic nerve. Optic atrophy may result from thick inflammatory exudate at the base of the brain surrounding the optic Chiasna and leading to vascular occlusion and fibrosis. It may also result from endarteritis obliterans, organisation of tubercles along the course of blood vessels, due to raised intracranial pressure leading to papilloedema and post neuitic atrophy, extension of surrounding neuroretinitis or by interstitial optic neuritis. The cause of papilloedema is attributed to the result of blockage of the inter meningeal spaces around the optic nerves by the inflammatory process.

Desai & Ankelsaria (1967) observed that the pathological basis of papillitis is opticochiasmatic arachnoiditis. It is due to further extension of meningitis along with the sheath of the optic nerve. The pale disc is suggestive of reduction in the blood supply whereas bitemporal pallor suggests a compression type of lesion at the level of the Chiasma.

Verma et al (1981) described that pupillary involvement in TBM may be due to the nerve involvement or due to pressure on the brain stem by dilation of third ventricle (Richman's theory). He further suggested that primary optic atrophy may be due to internal hydrocephalus of third ventricle causing pressure on optic chiasma which in turn causes primary optic atrophy.

DIAGNOSIS

The diagnosis of TBM is based on certain supportive and diagnostic criteria. The supportive tests primarily include a positive tuberculin skin test and an X-ray chest showing a primary focus. Lincoln (1947) and Lincoln & Sowell(1963) studied the incidence of positive montoux test in cases of tuberculous meningitis. In both the studies authors found that montoux test was positive in about 85% of cases of tuberculous meningitis. The diagnosis criteria includes a CSF examination. Heinz and Thomas (1979) have reported that white blood cells in CSF are usually fewer than 350/cubic mm and consists mainly of mononuclear cells, but in few cases the fluid showed upto 1000 cells, with a predominence of polymorphonuclear cells. Wright (1953) showed that by the time the disease was well established the sugar was usually below 45mg/100 mg and thought in the early stages protein concentration was normal or slightly elevated but with time it is increased to 300 mg/100 ml or more. Gierson and Marx (1955) studied the significance of chloride estimation in the diagnosis of TBM. These authors reported that the chloride content almost always falls below 115 meq. Thaper (1967) observed that isolation of tubercle bacilli from the CSF varies with different techniques

3% by ordinary centrifuge method, 38.8% by precipitation of deposit from CSF by alcohol and 79% by floatation hydrocarbon technique.

Gupta & Chopara (1981) in their review article mentioned minimum diagnostic criteria for TBM. They reported that the CSF examination revealing raised proteins with low sugar and pleocytosis predominantly of lymphocytes along with the history and clinical picture co-firms the diagnosis of TBM. They also mentioned tests, viz. Nitroblue Tetra zolium test, Bromide partition test, Lactate Dehydrogenase Isoenzymes and cerebrospinal fluid protein-ogram, which have been used and found useful in TBM.

OPHTHALMIC MANIFESTATIONS

Tooke (1915) was perhaps the first worker to have done an extensive study on cases of tuberculous meningitis with regard to the various ophthalmological manifestation. He studied 70 cases of TBM and observed that the fundoscopic findings were mainly optic neuritis in 43% of cases, while only one case had presence of papilloedema.

Illingworth and wright (1948) in their study of tuberculous meningitis and miliary tuberculous reported the presence of choroid tubercles. They divided these cases in three groups. In first group composed of 18 cases of TBM without millary tuberculosis. In second group there were 28 patients of TBM with millary tuberculosis while in third group 14 patients of miliary tuberculosis without TBM were

studied. The author observed the presence of choroid tubercles in the three groups to the extent of 15.5%, 64% and 50% respectively.

Mager (1949) has drawn attention to the optic nerve involvement in his study of tuberculous meningitis. He reported optic nerve involvement in 33% of cases. He commented that swelling of the papilla in the initial stages of the disease as being of no importance with regard to diagnosis or prognosis while complication may be expected in the presence of choked disc.

Isente Ivan (1950) studied 300 cases of tuberculous meningitis. He reported in his study a high incidence of ocular involvement in the form of chorioretinitis and disc changes in 95% cases. Dolcet et al (1950) studied 63 cases of tuberculous meningitis. The workers also reported optic nerve involvement in majority of their studied cases.

Arouth & Zanbarow (1953) carried out yet another study in 100 cases of TM. to evaluate the various ophthalmological changes. He observed that out of the 100 children studied 45 cases exhibited chorioretinal lesions, 41 showed alterations of the optic nerve head in the form of 30 cases of active papilloedema, 8 cases of passive papilloedema and 3 cases of optic atrophy. The authors however failed to report any choroid tubercle in their study.

Mooney (1956) in his extensive study of 15 months, studied 65 cases of tuberculous meningitis for

various ocular sequelae of the 65 patients examined 6 died from meningitis. Out of these 65 cases 47 cases (72%) had ocular lesions, some of which were multiple. The author observed papilloedema in 26% of cases, pale discs in 20% and retrobular neuritis in 3.1% cases. Ocular palsies were noted in 15.5% cases where as choroid tubercles were reported in 25% of cases.

Laha & Dev (1956) studied 32 cases of tuberculous meningitis for various clinical manifestations of disease. They observed optic nerve involvement in 18.8% of their cases mainly in the form of optic atrophy.

Lincoln and Sordillo (1960) studied 241 cases of tuberculous meningitis and evaluated mainly the extra ocular changes in their study. They divided the cases into the two groups, according to whether they were treated (74 cases) or untreated (167 cases). The authors reported that in both the groups facial palsy was present in 7 cases, while ptosis was observed in 7 untreated cases as compared to only one case in the treated group. Similarily the authors observed a higher incidence of strabismus in untreated (16 cases) than the treated group (10 cases).

Khatua (1961) studied 231 cases for extensive clinical study of tuberculous meningitis. He observed pupillary abnormalities in majority of his studied cases. The incidence of pupillary abnormalities was 76% in his study group.

Bhatnagar & Srivastava (1961) carried out an study to evaluate the various ocular changes in tuberculous meningitis in children. The clinical material comprised of 30 patients, who were admitted in the children medical wards of Hamidia Hospital, Bhopal from March 1960 to February 1961. A detailed clinical examination was done and a thorough ophthalmic check up carried out on admission and repeated at weekly intervals. The various ocular manifestations included pupillary changes in 50% cases in the form of dilated & fixed pupil, 16.6%, moderately dilated with sluggish reaction 20% and an isocaria 13.3%, conjugate deviation of eye balls 13.3%; changes in eye grounds in 50% of the cases. Optic nerve changes were found in 43.3% of the patients of whom 13.3% had optic atrophy and 16.6% exhibited papilloedema. Choroid tubercles and choroiditis were manifested in 3.3% patients each. Ocular muscle palsies were observed as 16.6% III nerve palsy and 6.6% VI nerve palsy. In their study 33.5% patients had no ocular lesions.

Dutta (1962) studied 2462 children for 'pattern of eye disease in children '. In his studied cases he reported 28 cases of optic atrophy. The common cause attributed to optic atrophy in his study was tuberculous meningitis.

Gupta & Webb (1962) studied tuberculous meningitis in 117 children of south Indian zone to evaluate the clinical manifestations of the disease. They observed pupillary abnormalities in 35% of their cases. The cranial nerve

palsies involving third, sixth and seventh were observed in 15.4% cases.

Miller (1962) studied 38 patients of tuberculous meningitis. He reported 2.7% cranial nerve palsies while analysing the various sequelae of the disease.

Misra and Gupta (1962) carried out another study for ocular complications of tuberculous meningitis they studied 40 patients of TBM for various ocular involvement. They reported chorioretinitis in 7.5% cases. The optic nerve involvement was 57.5% in the form of papillitis 35%, papilloedema 7.5% retrobular neuritis 5% and optic atrophy 29.5%. 20% cases of papillitis, 5% of Papilloedema and 15% optic atrophy recovered. The ocular palsies were observed in 30% cases. The oculomotor nerve involvement was present in 12.5% of cases of whom 5% was totally and 7.5% was partially involved. The abducent and facial nerves were involved in 17.5% and 7.5% cases respectively. The workers also observed pupillary abnormalities in 25% cases of which 10% cases were having dilated and fixed pupil, 7.5% sluggish and 7.5% anisocoria (1962).

Bharucha & Talwarkar (1962) in their study for treatment of tuberculous meningitis observed pupillary abnormalities in 24.99% of cases in the form of dilated and fixed pupil.

Verma and Agarwal (1966) carried out an extensive study for ocular manifestations of tuberculous

meningitis in children. The cases were below age of 16 years and were selected from TB patients of S.R.N. Hospital and M.L.N. Medical College, Allahabad. They included 65 cases for the study group. The optic nerve changes observed were papillitis 12.3%, papilloedema 7.6% and optic atrophy 9.2%. The pupillary abnormalities reported were 24.6% in the form of dilated and fixed pupil. The cranial nerves involved in the study were facial in 10.7% cases, abducent in 9.2% cases and oculomotor in 3% cases. The workers also observed miliary tubercle of choroid in 1.5% cases.

Nanchanda & Lal (1966) studied 249 complicated cases, of TBM out of 742 children suffering from tuberculosis in V.J. hospital Amritsar. They carried out an extensive study for various problems of tuberculous meningitis. The cranial nerve involvement in their cases was having a very low incidence. They observed only 1 case of facial palsy. Optic atrophy was reported only in 6 cases of the total studied cases of TBM.

Desai and Ankelsaria (1967) studied 47 cases of tuberculous meningitis with special reference to the ocular aspect. Out of these 37 were from the paediatric and 10 were from other medical wards. Optic nerve affection was seen in 74.46% cases, other cranial nerve involvement was in 25.53% cases and pupillary changes in 63.82% cases. They observed 6th nerve palsy in 14.09% cases out of them in followup study

1 case left against medical advice and others recovered. 3rd nerve palsy was seen in the form of ptosis out of them 50% recovered completely and 50% partially. Seventh nerve palsy was seen in 8.5% cases and all cases recovered completely. Posterior segment changes were found with greater frequency. Pale disc was observed in 12.76% cases out of them 50% remained same, 33.33% progressed to optic atrophy and 16.66% cases expired infollow up study. Papilloedema was observed in 10.63% cases out of them 50% cases cleared up, 25% progressed to optic atrophy and 25% expired. Papillitis was seen in 29.78% cases of them 69.2% cleared, 7.69% progressed to optic atrophy and 23% cases expired. Optic atrophy was seen in 14.89% cases out of which 85.7% cases remained same in follow up study and 14.2% expired. Retrobulbar neuritis was observed in 2.1% cases which all recovered. Bitemporal pallor was reported in 4.2% cases of them 50% cleared and 50% remained same. The workers were not able to report choroid tubercles in their study.

Thapar et al (1968) carried out a study to report ocular manifestations in tuberculous meningitis. They studied 100 cases of tuberculous meningitis from those admitted to the department of paediatrics S.N.Medical College, Agra in years 1965-66. Optic nerve involvement was present in 49%, pupillary abnormality in 12% and cranial nerve palsy in 17% cases. The fundus examination revealed papillitis 25%, papilloedema 13% and optic atrophy 11%. The pupillary abnor-

mality observed was in 12% cases in the form of dilated and fixed pupil. The various cranial nerve palsies were VIIth nerve in 8%, IIIrd in 4% & VIth in 5% cases. The workers also observed nystargmus in 3% cases.

Agarwal and Kumar (1969) in follow up study of treated cases of tuberculous meningitis studied 25 cases. The cases were followed up from 6 months to 3 years. They observed and incidence of 12% blindness. The optic nerve involvement was seen in 44% cases. 32% cases were having visual impairment. They also reported 7th cranial nerve palsy in 28% cases.

Rama Chandran et al (1970) studied 288 children of TBM selected from 2554 cases of tuberculous patients below 12 years registered in the tuberculosis clinic of Raja Mirasdar and Thanjavur. Medical College Hospital, Thanjavur, Tamilnadu for the period from 1964 to 1969. They observed blindness in 46% cases as residual sequelae. Cranial nerves involvement was also observed. Facial nerve was involved in 27 cases where as IIIrd and VIth nerve involvment was reported in 1.8% cases each.

Saxena & Tomar (1970) carried out an extensive study of 100 patients of TBM for ocular lesions especially. Various ocular manifestations were in 89% of studied cases, 11% being normal. Fundus findings were seen in 55% cases. These included papillitis in 56.4%, papilloedema in 12.7%,

optic atrolhy in 29.1% and choroid tubercales in 1.8% cases. The workers observed dilated and fixed pupil in 53% cases cranial nerve palsies were seen in 47% cases out of which VIIth nerve was involved in 23.5% cases where as IIIrd and VIth nerves were involved in 11.7% cases each.

Lothe et al (1972) studied 91 patients of tuberculous meningitis with special reference to complications of the disease. They observed pupillary abnormalities in 80% cases. The cranial nerve palsy was seen in 32.2% cases. They reported optic atrophy in 67.7% cases where as loss of vision was observed in 8% cases.

Udani et al (1974) studied 500 cases of tuberculosis of central nervous system. In their study they also observed for involvement of cranial nerves. Involvement of 2nd nerve was observed in 14.2% cases where as third ,Sixth and seventh nerves were involved in 5.4%, 10.8% and 29% cases.

Benakappa et al (1975) carried out an study of 50 cases of TBM among children who were admitted to the paediatric ward of Vanivilas Hospital Banglore. They noted fundus changes in 24 % cases. Cranial nerve involvement was observed in the form of sixth cranial nerve (20%) and facial nerve(14%).

Smith (1975) made an attempt to study the ocular changes in tuberculous meningitis in children. He observed pupillary abnormalities in 48.2% cases. Various cranial nerve palsies involving 3rd, 6th and 7th were seen in 13% of his cases.

Tamaskar and Bhandari (1976) in their clinicopathological study of meningitis in infancy and childhood studied 50 cases. Out of 50 cases 29 cases were of tuberculous meningitis. The fundus changes were seen in 45% cases in the form of optic atrophy and papilloedema cranial nerve palsies were reported in 14.3% cases.

Idriss & Sinoo (1976) studied 43 children suffering from TBM who were admitted to the American University Medical Centre of Beirut. They observed uptic atrophy only in 9% cases. Various cranial nerve palsies were reported in the form of third nerve, sixth nerve and seventh nerve each in 9% cases.

Gupta (1976) studied 50 cases of tuberculous meningitis to evaluate the optic nerve involvement. Out of 50 cases, 25 cases were taken from children ward of civil hospital, Jhansi. Optic nerve involvement was observed in 88% cases. The optic nerve was involved in the form of temporal pallor in 20%, papilloedema in 12%, primary incomplete optic atrophy in 20% and primary complete optic atrophy in 36% cases. It was interesting to note that no case was reported having post neuritic atrophy. Pupillary abnormality was observed in the form of sluggish reaction in 32% cases.

Verma et al (1981) carried out an recent study regarding various, ocular manifestations of tuberculous meningitis and their prognostic value in children. A total

50 cases were selected for this study. Ocular involvement was observed in 70% of total cases. The workers observed conjunctivitis and corneal ulcer in 6% cases each in their study. They have reported pupillary abnormalities in 40% cases. 8 cases showed dilated & fixed pupil, 6 cases semi-dilated pupil, 4 cases sluggish pupil and 2 cases anisocoria. It was evident that mortality was highest in cases with dilated & fixed pupil i.e. 25%. Cranial nerve involvement was a common feature and in this series the acculomotor nerve involvement was seen in 12% of cases, 2 cases showed a complete third nerve involvement while 4 patients showed partial third nerve involvement. Complete involvement showed mortality 16.6% while there was no mortality in partial third nerve involvement. Abducent nerve was also involved in 12% cases 10% cases had unilateral involvement. The patients having abducent nerve palsy showed a mortality of 33.3%. Facial nerve was involved only in 2 cases. 31 cases had optic nerve involvement in this series. Primary optic atrophy was seen in 20% cases, whereas secondary optic atrophy was observed in 12% cases. It was interesting to note that there was no mortality in patients having primary optic atrophy. Temporal pallor was seen in 5 cases Papilloedema was seen in 4 cases, out of which 3 expired thus showing grave prognosis. 6 cases exhibited papillitis out of 6, 2 cases expired.

MANAGEMENT

Steroid :

The workers viz. Bulkley (1953), A shby & Grant (1955), Koshy et al (1955), Dhir et al (1959), O'Toole et al (1959), Misra & Khanna (1961), Athavale (1961) Smith (1963), Hockaday & Smith (1966), Kapur (1969), Rama Chandran et al (1970), Gordon & Parson (1972), Udani et al (1974), Escobar et al (1975), Idris & Sinoo (1976), Kennedy & Fallon (1981), Jaffe (1982) and Meyers (1982) emphasized that steroids should be added to the therapeutic regimen in patients of tuberculous meningitis as adjunct to antitubercular treatment. These can be administered by various routes like oral, intramuscular, intravenous, intrathecal or retroocular. Besides improving survival rate cortico-steroids play definite role in the treatment of cerebral oedema reducing spinal block & depressing the tuberculin hypersenstivity.

Steroids in optic nerve involvement-Boyd (1945) described that early detection & judicious therapy instituted at the stage of optic neuritis could prevent occurence of optic atrophy in most of the cases.

Bhatnagar & Srivastava (1961) observed that treatment of optic atrophy lies in its prevention. Many exudatives agents and cortisones were tried for, workers in the field, in treatment of optic atrophy but all except cortisones failed to stand the test of time.

HYPEROSMOLAR AGENTS :-

Udani et al (1974) observed that Glycerol 10% in a dose of 1.5 to 2 gm/Kg. Orally or intravenously may help to reduce the oedema over a long period. Mannitol (100 to 200 ml) intravenously twice a day should be tried to reduce the oedema. Gupta & Chopra (1981) described that apart from cortisones, glycerol (0.5-1.5 gm/kg.) body weight) every 4-6 hourly intravenously or orally is useful for chronic treatment of raised intracranial tension. The most frequently used drug is, mannitol (20%, 1.5 - 2 gm/Kg. body weight) by intravenous route, in the management of oedema.

Vasodilators - Keith Lyle (May & worths manual disease of eye, 3rd ed. 1968) described that vasodilators are useful in the dilation of occluded central artery of retina or its branches. In an inadequate supply to retina we can administer tolazoline hydrochloride 2 mg/ thrice daily orally. It can also be injected retro-ocularly.

RETROBULBAR INJECTIONS

Duke Elder (system of ophthalmology Vol. VII, 517, 1971) have given a description of retroocular treatment. In this volume he has mentioned that the technique was introduced by weiss & was standarized by lowenstein. In this technique a long fine needle is inserted into the lower lid at the inferotemporal angle of the orbit so as just to clear the orbital margin and is pushed inwards, medially and slightly

upwards some 3.5 cm in the direction of optic foremen. It is well to aspirate to eliminate the very unlikely point of the needle entering an abnormally large vein. Attempts have been made to introduce the medicaments into the posterior segment of the globe or to reach the optic nerve by this route. Further conditions in which such injections have been employed include the use of vaso-dilators in case of occlusion of the retinal artery and medicaments in optic atrophy.

MATERIAL AND METHODS

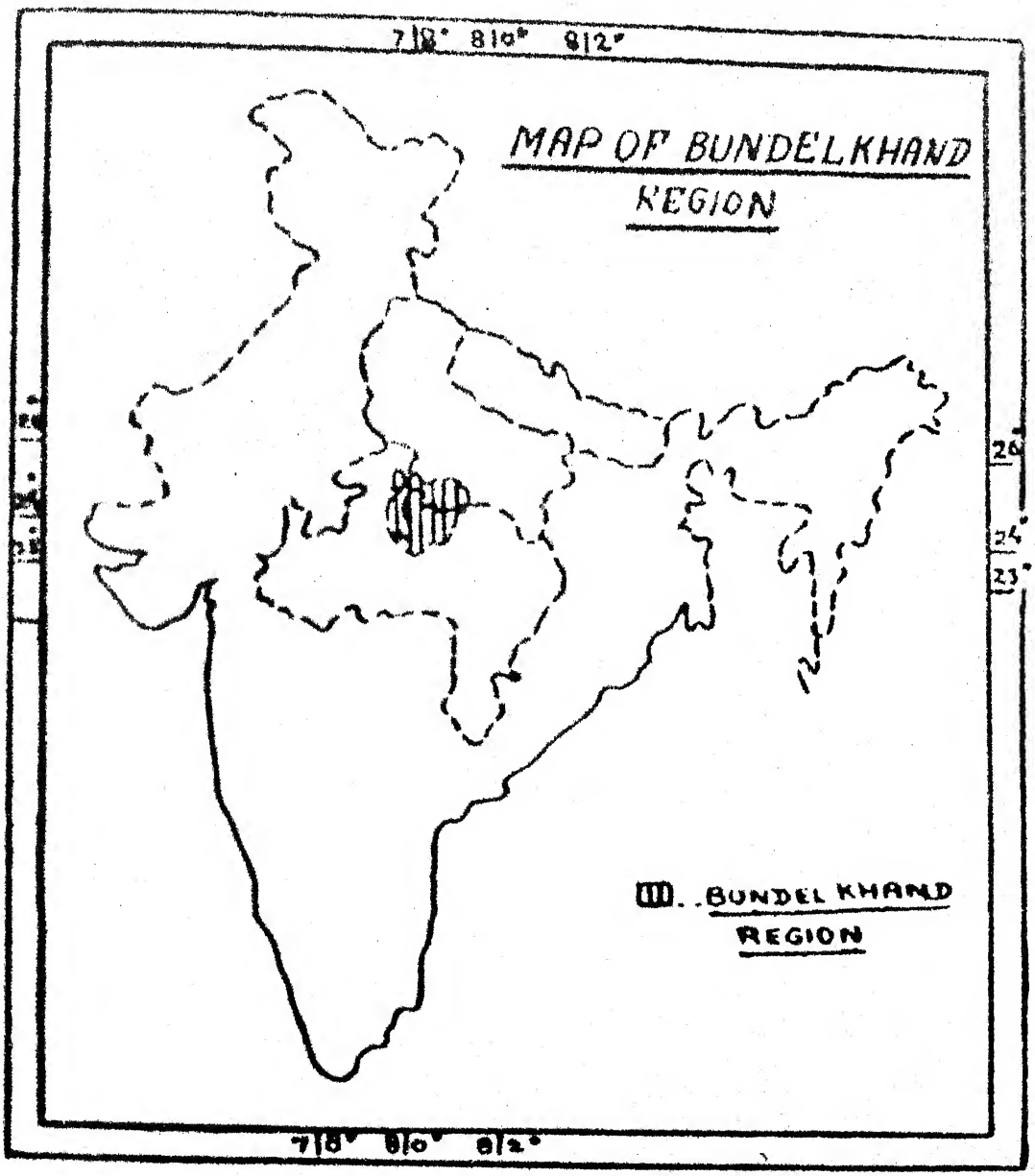


Fig. 1

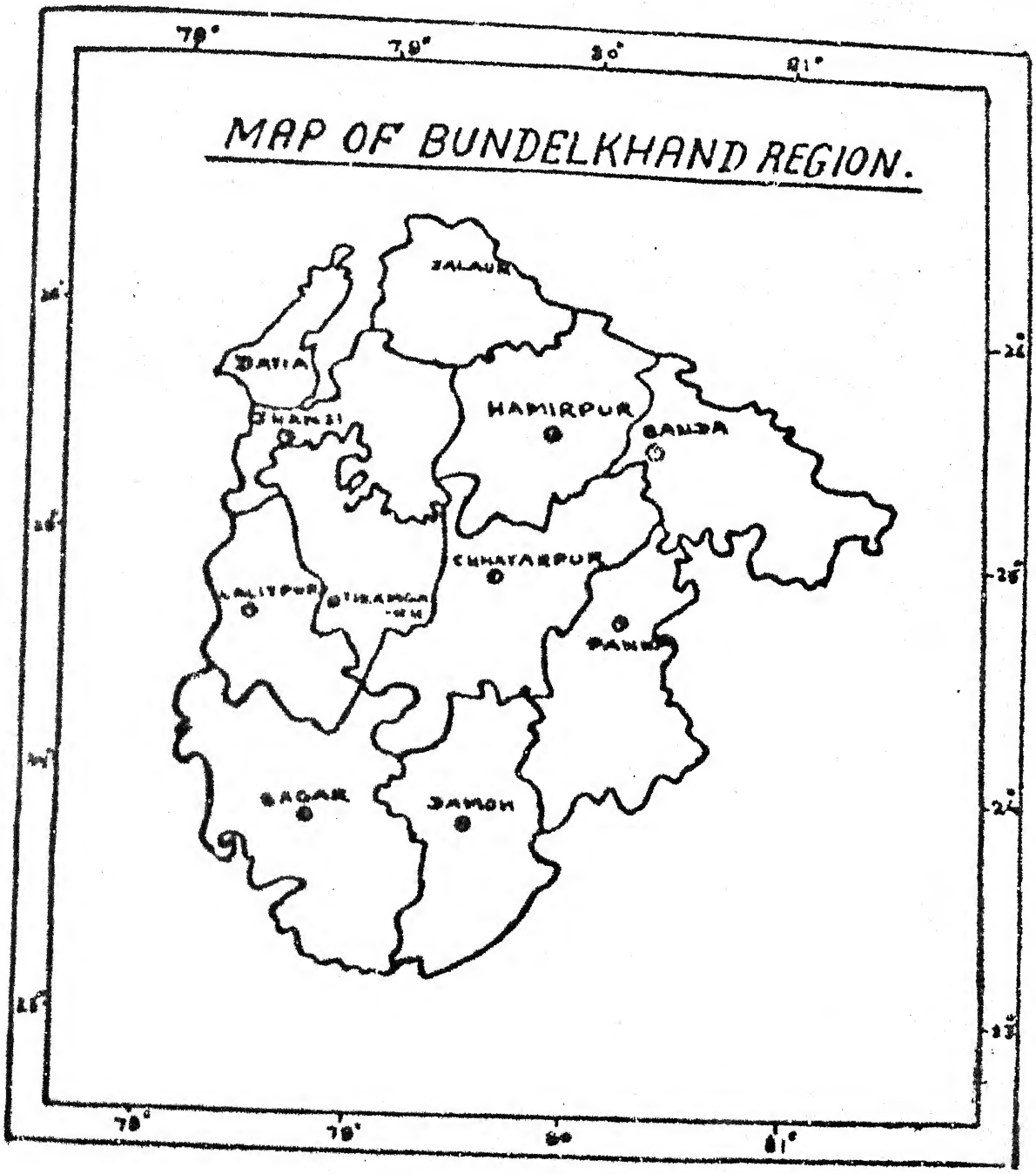

MAP OF BUNDELKHAND REGION.
JALAUN
DATIA
JHANSI
HAMIRPUR
BANDA
CHHATARPUR
LALITPUR
SAGAR
DAMOH
78°
79°
80°
81°

Fig. 2.

MATERIAL AND METHODS

The present study was carried out in the department of Ophthalmology, M.L.B. Medical College, Jhansi in active collaboration with the department of Paediatrics, over a period of 11 month from July, 1983 to May, 1984. Children, admitted to the paediatric ward, M.L.B.Medical College,Jhansi and diagnosed as cases of tuberculars meningitis were selected for this study. Children were of either sex and the age ranged from under 1 year to 13 years.

Sixty three children of tuberculous meningitis were studied for ocular manifestation and their management. These children admitted to the Paediatric ward of M.L.B. Medical College, Jhansi, belonged to Jhansi district and nearby districts of Uttar Pradesh and Madhya Pradesh forming the Bundelkhand Region.

The area of study, Bundelkhand Region which spreads over the states of Uttar Pradesh and Madhya Pradesh (Fig. 1), is located between 23°10' and 26°30' North Lattitude and 78°21' and 81°40' East longitude. The region covers a total geographical area of 7000 Sq. Km. including eleven districts, Five of which viz. Jhansi, Lalitpur, Jalaun, Hamirpur and Banda are in Uttar Pradesh and remaining six districts viz. Datia, Tikamgarh, Chhatarpur, Panna, Damoh and Sagar in Madhya Pradesh (Fig. 2).

The region represents a transitional zone of tropical dry subhumid in the east to tropical semiarid in the west. The overall mean annual temperature of the region is high and varies from 25 - 26°C.

METHOD :

Diagnosis of TBM :

The diagnosis of tuberculous meningitis in these children was based on clinical features and was confirmed by cerebrospinal fluid examination. Cerebrospiral fluid examination revealing increase in protein, moderate decrease in sugar content and pleocytosis varying from 25 to 500 cells per cumm, the predominent cell type being lymphocytes confirmed the diagnosis.

Examination of Child :

A detailed history of present illness pertaining to an insidious onset of fever, vomiting, headache in an older child, change in disposition of the child, convulsions and alteration in consciousness, was recorded in each case.

Due importance was given to obtain the history of contact in each case, as well as to record the immunisation status regarding B.C.G. vaccination. The nutritional status of the child was evaluated by a detailed dietary history. Past history was recorded in each case to elicit a definite history of primary complex, measles, pertussis, ear discharge, head injury, convulsions and worm infestations.

A detailed clinical examination was carried out to assess the stage of the disease by a thorough neurological examination. In general the pattern in the untreated child may be divided into 3 stages (according to WALDOE, NELSON, Text book of Paediatrics).

I. Prodromal stage - In this stage the manifestations are vague. There may be fever, change in disposition, irritability, complaints of headache, anorexia, vomiting and constipation.

II. Transitional stage - convulsions, meningeal signs, exaggeration of deep reflexes, ocular paralysis and vasomotor disturbances are the main manifestations of this stage. In this stage course is progressive and the drowsiness tends to be replaced by stupor.

III. Terminal stage - comatose stage, dilated and unresponsive pupils, wide spread paralysis, irregular pulse and irregular respiration and raised temperature are the important findings of this stage.

Ocular examination :

A detailed ocular examination was carried out in each case of tuberculous meningitis under following heads -

(1) Ocular complaints - regarding pain, redness, watering discharge, unable to identify objects or deviation of eyes were noted.

(2) Ocular aspect - Due importance was given to the pupillary examination. Presence of ptosis, nystagmus or squint were

noted. Involvement of cranial nerves especially of IIIrd, VIth and VIIth were also noted. A detailed ocular examination was carried out with help of Binoocular loupe and bright torch light.

(3) Ophthalmoscopy - A detailed fundus examination regarding media, optic disc (size, shape, colour, margins and cup) retinal vessels, macula, choroid and retina, was carried out in each case of tuberculous meningitis by Keeler's, Direct Ophthalmoscope. The dilatation of pupil was carried with 10% phenyl ephrine or atropine 1% and child was under sedation during fundus examination.

First ophthalmoscopic examination was carried out at the time of admission on the basis of provisional diagnosis of tuberculous meningitis based on clinical picture. Lateron when the diagnosis was confirmed repeated fundus examination was carried out in each case, initially biweekly and later on weekly during the hospital stay of the child. The cases having ocular problems were followed up after discharge from hospital for management.

The optic nerve involvement was noted in the form of papillitis, papilloedema or optic atrophy. The criteria for diagnosing papillitis, papilloedema or optic atrophy was based on Duke Elder's description (System of Ophthalmology Vol XII - Neuroophthalmology 1971).

I. Papillitis (Fig. 3) - The diagnosis of papillitis based on following points.

- Disc hyperaemic with blurred margins.
- Exudates and haemorrhages may be present in the tissue of disc but in less quantity.
- Pain or tenderness on movement of eyewall espeicially upwards and medially.
- Sudden visual loss.
- Defective pupillary reaction to light.

II.Papilloedema (Fig. 4).

- Increased redness of the disc with a slight haziness and blurring of its margins first at upper and lower nasal quadrants and lateron round the disc.
- At fully developed papilloedema the disc assumes the color of the retina and its position is indicated only by the confluence of the larger vessels small linear haemorrhages and exudative patches are present and oedema is more than 2 dioptres.

III-Optic atrophy (Fig. 5)- The diagnosis of optic atrophy was based on the visual acuity of Child, pupillary reactions and colour of disc.

From point of ophthalmoscopic appearances optic atrophy grouped into 2 types in this study.

(a) Primary - The disc is white with sharply defined edges.

- Size diminished and having a saucer shaped excavation.
- Minute vessels of disc disappeared.
- Surrounding retina having usual appearance.
- Retinal vessels appearing normally but arteries are having diminished calibre.

(b) Post Neuritic - Disc dense white or greyish with irregular margins.

- Haze present, minute vessels lost and it is covered by connective tissue.
- Lamina cribrosa is hidden.
- Retinal vessels enclosed by white lines.

Investigations of case - Each case of tuberculours meningitis was investigated for leucocyte count (total and differential) haemoglobin, erythrocyte sedimentation rate and other specific necessary investigations if needed.

Management of ocular problems :

Antitubercular treatment (Isoniazid, Streptomycin, Ethambutol, and Rifampicin) was given in each case along with supportive therapy. Cortisones were added to the antitubercular treatment as an adjuvant. These were administered by different routes viz. Orally, intramiscularly, intravenously or by retrobular injections.

The management of ocular problems was carried out as follows -

Ocular problems - To prevent the eye from exposure keratitis and corneal involvement in various ocular pareses, Achromycin

eye suspension was applied locally. In cases of corneal ulcer local application of antibiotic ointment along with atropine eye ointment was given with pad & bandage.

Papilloedema - In the management of lowering intracranial tension following drugas were given according to the merits of case.

1. Mannitol (20%) 1.5 - 2 gm/kg. body weight was administered 6 hourly by intravenous drip.
2. Acetazolamide 25-30 mg(Kg. body weight given orally in 2-3 divided doses.
3. Glycerol 0.5 - 1.5 gm/kg. body weight was given orally in 2-3 divided doses.

Papillitis and postneuritic atrophy -

In these cases along with dexamethasone (Decadron), Vasodilators viz. tolazoline hydrochloride (Priscol) were given by R/B injections. A course of 10 injections one on alternate day was given in the dose of 1 mg dexamethasone and 6.5 mg tolazoline hydrochloride to each eye.

B_1, B_6 and B_{12} (Trioredisol drops) and xantinolnicotinate (Complamina) 75 mg thrice daily were given minimum for 2 months and more depending on the condition by systemic route.

Fig - 3 : PAPILLITIS

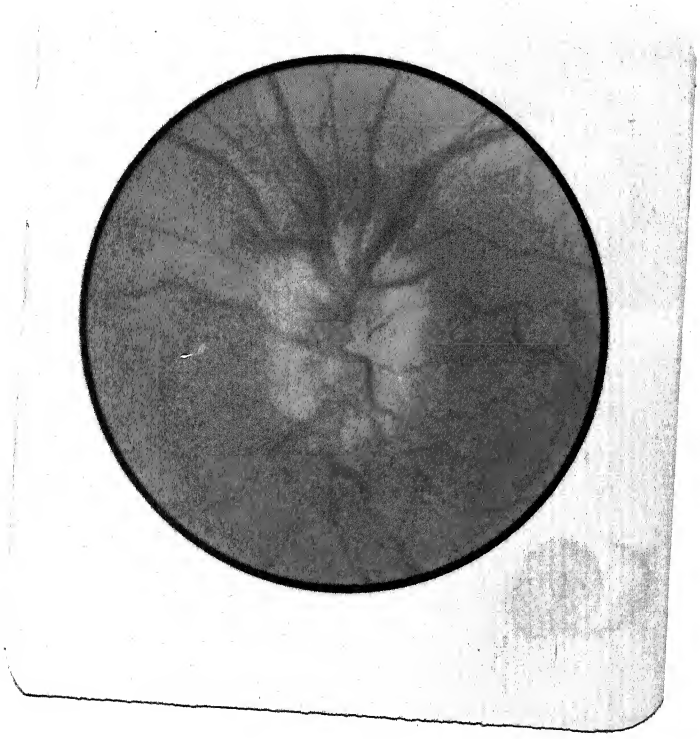
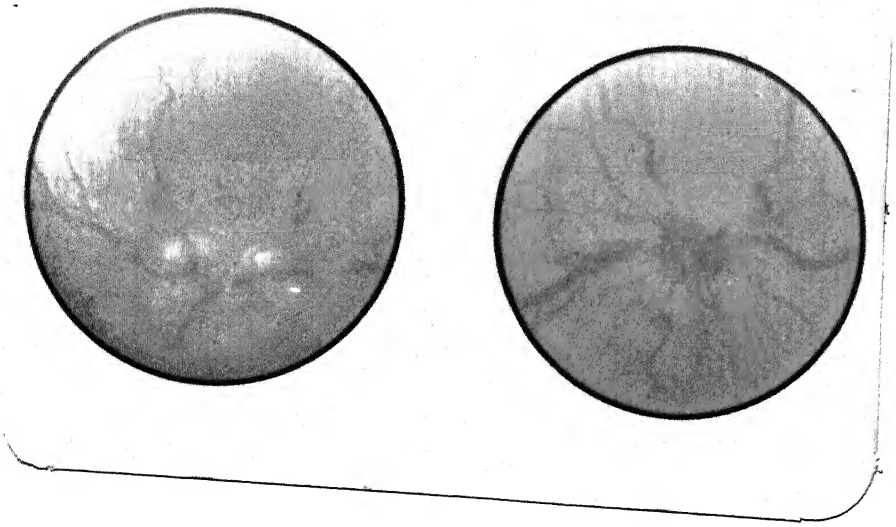


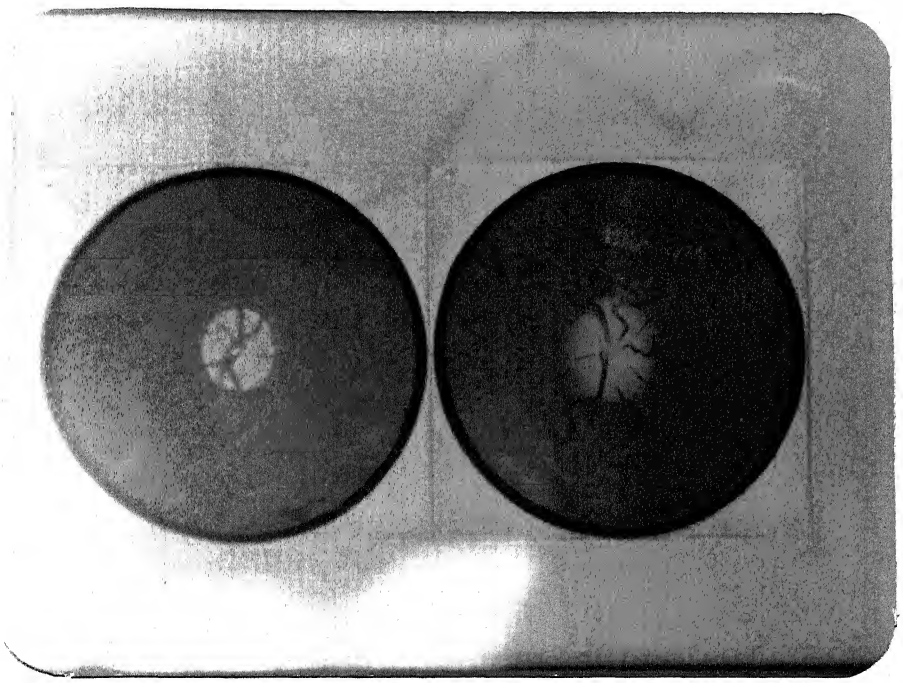
Fig - 4 : PAPILLOEDEMA



Early Stage

Late Stage

Fig - 5 : OPTIC ATROPHY



Primary

Post-neuritic

OBSERVATIONS

OBSERVATIONS

The present study was carried out in 63 tuberculous meningitis (TBM) patients (Upto the age of 13 years) in the Department of Ophthalmology in active collaboration with Department of Paediatrics, M.L.B.Medical College, Jhansi. All the 63 cases suffering from TBM were carefully examined for ocular problems. Out of total 63 patients, 54 (85.71%) cases were observed to have ocular involvement.

TABLE NO. I

DISTRIBUTION OF PATIENTS OF TBM IN DIFFERENT AGE & SEX GROUPS

Age groups (years)	Sex distribution		Total No.	Percent
	Male No.	Female No.		
0-2	25	8	33	52.38
2-4	15	3	18	28.57
4-6	3	1	4	6.34
6-8	2	-	2	3.17
8-10	3	-	3	4.76
10-12	2	-	2	3.17
12-14	1	-	1	1.58

It is evident from table I that maximum number of children suffering from TBM were in the age groups of 0-4 years of age which constituted 80.95% of the total cases in our study. It is also clear that there was preponderance of male cases over female. The total female cases were only 19.04% (Fig.1).

BAR DIAGRAM SHOWING AGE & SEX INCIDENCE IN T.B.M.

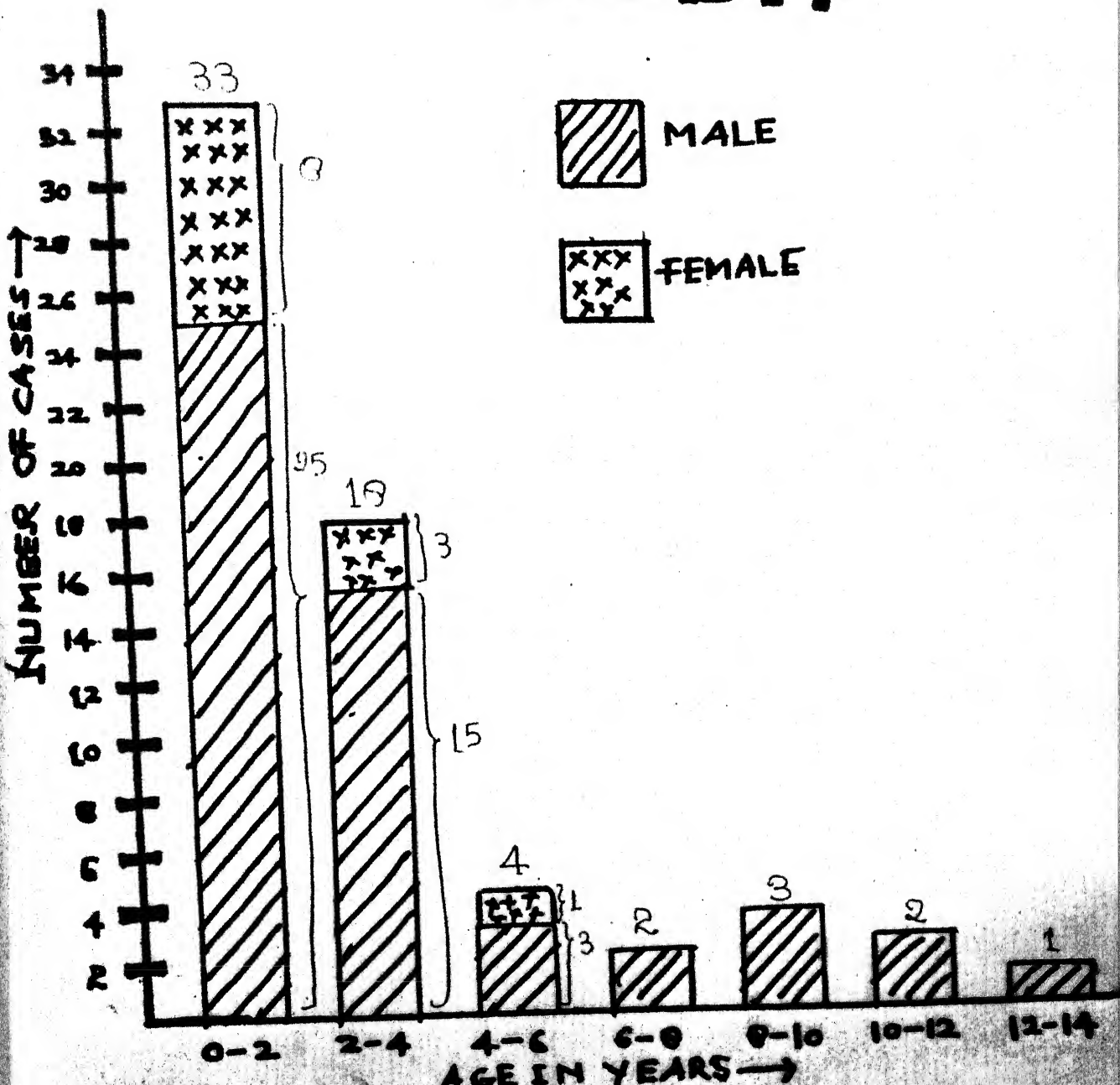
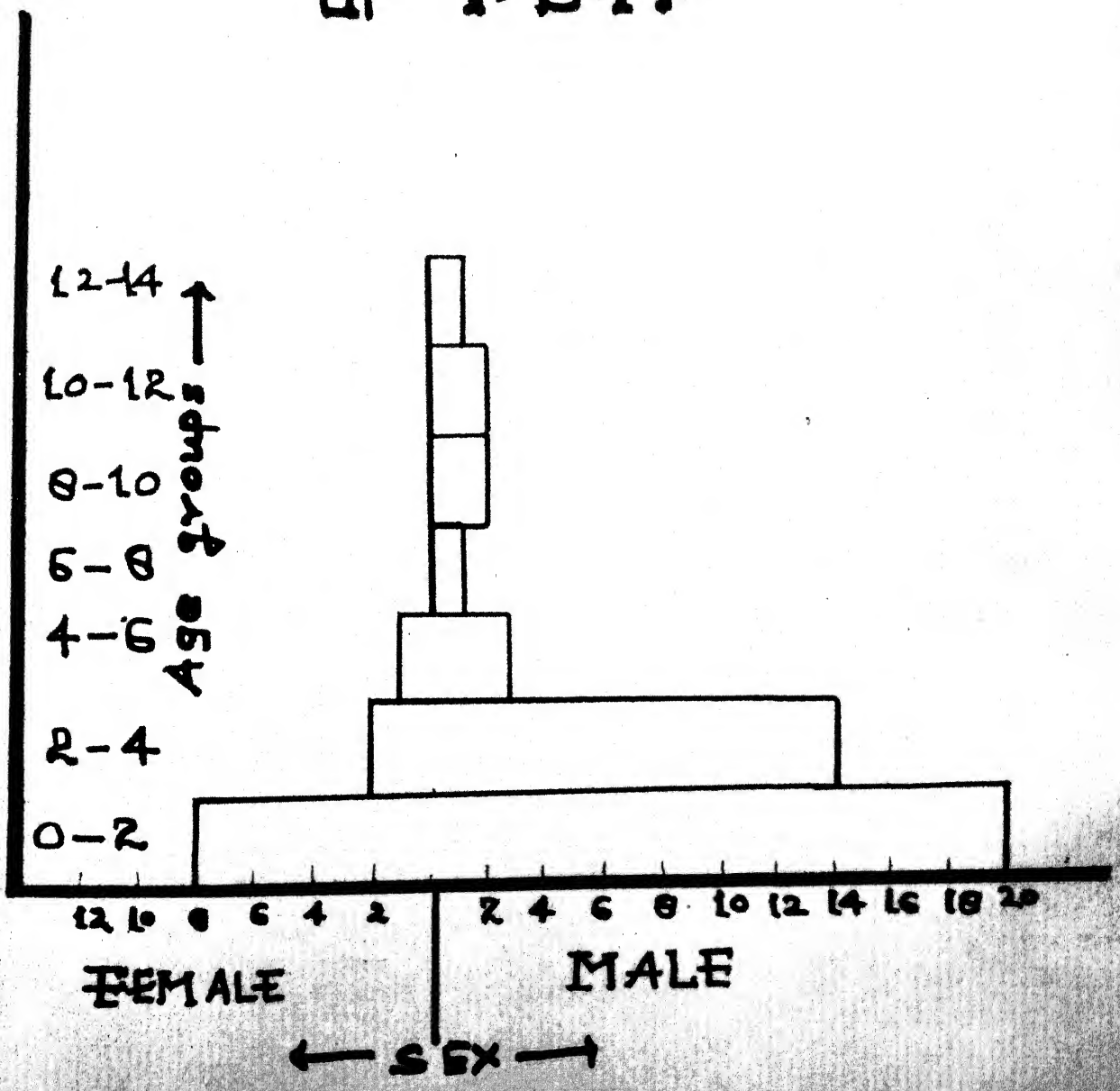


Fig-1

Fig-2

PYRAMID DIAGRAM SHOWING AGE & SEX DISTRIBUTION IN OCULAR INVOLVED CASES OF T.B.M.



Distribution of various signs & symptoms in TBM is shown in Table III.

TABLE III

DISTRIBUTION OF VARIOUS SIGNS & SYMPTOMS IN TBM

Signs & Symptoms	No.of cases	Percent
Fever	54	85.71
Vomiting	37	58.73
Convulsions	37	58.73
Altered sensorium	52	82.53
Meningeal signs	48	76.19
Hemiparesis	7	11.11
Quadriparesis	4	6.34
Monoparesis	3	4.76
Hemiplegia	3	4.76
Cough	6	9.52
Diarrhoea	7	11.11
Constipation	1	1.58
Pain in abdomen	2	3.17
Headache	2	3.17
Hepatomegaly	7	11.11

It is clear from table III that the most pronounced signs & symptoms in the series were fever (85.71%), vomiting (58.73%), convulsions (58.73%), altered sensorium (82.53%) and meningeal signs (76.19%).

Table IV shows prevalence of ocular lesions and deaths in 63 paediatric cases of TBM in Bundelkhand region.

TABLE IV

PREVALENCE OF OCULAR LESIONS & DEATHS IN TBM PATIENTS

Patients of TBM	Prevalence		Deaths	
	No.	%	No.	%
With ocular lesions	54	85.71	16	25.39
Without ocular lesions	9	14.29	-	-

It is evident from table IV that the TBM cases of Bundelkhand region showed a high prevalence (85.71%) of ocular involvement. The mortality observed was 25.39%. It is interesting to note that all the patients showing mortality were exhibiting ocular problems.

Relationship of ocular involvement with various factors viz. socioeconomic status, nutritional status, stage of disease and duration of disease were studied and presented in details.

Socio-economic status - Table V shows that majority of patients under study were in low socio-economic status belonging to socio-economic groups III & IV & V. Only 7 cases were from socio-economic group II and none from group I.

TABLE V

PREVALENCE OF OCULAR PROBLEMS IN TBM BY SOCIAL CLASS.

Mean monthly percapital income	Social class	Total patients		Mortality		With ocular problems		without ocular problems	
		No.	%	No.	%	No.	%	No.	%
Rs. 600 & Above	I	-		-		-		-	
Rs. 300 - 599	II	7	11.11	1	14.28	3	42.85	4	57.15
Rs. 140 - 299	III	18	28.57	3	16.66	15	83.33	3	16.67
Rs. 60 - 139	IV	23	36.50	8	34.78	21	93.30	2	6.70
Rs. ⟨ 60	V	15	23.81	4	26.66	15	100	-	-

Notes- Social classification according to Shrivastava et al (1981).

Analysis of table V reveals involvement of eye in TBM belonging to poor socio-economic groups - viz 88.33%, 93.30% and 100% patients to groups III, IV & V respectively. In group II the involvement observed was 42.69%. It is also evident from table V that mortality was more in class IV (34.78%) and class V (26.66%).

Nutritional status -

Table VI shows distribution of patients according to nutritional status. It was observed that majority of the studied patients were having poor nutritional status. Only 10 cases showed fair nutritional status and out of these 10 cases, 4 cases were having ocular involvement.

TABLE VI

PREVALENCE OF OCULAR PROBLEMS & DEATHS IN TBM BY NUTRITIONAL STATUS.

Nutritional status /75% expected wt. of Harvard standard (poor nutrition)	Total patients		Deaths		With ocular problems		Without ocular problems	
	No.	%	No.	%	No.	%	No.	%
Fair	10	15.87	-	-	4	40	6	60
Poor	53	84.13	16	25.39	50	94.33	3	5.66

t 6L. d.f. = 5.4

p / 0.001 (Highly significant)

It is evident from table VI that patients having fair nutrition showed ocular lesions prevalence (40%) where as in the patients having poor nutritional status prevalence was 94.33%. There was no death recorded in the patient having fair nutrition whereas mortality was 25.39% in patients of poor nutritional status.

Stage of disease - Table VII shows relationship of ocular involvement and mortality with stage of disease. It was observed that majority of TBM patients were in stage II & III and only 8 patients belonged to stage I.

TABLE VII

PREVALENCE OF OCULAR LESION & DEATHS IN VARIOUS STAGES OF TBM.

Stage according to Nelson (1975)	Total No.		Deaths		With Ocular problems		Without Ocular Problems	
	No.	%	No.	%	No.	%	No.	%
I	8	12.69	1	12.5	3	37.5	5	62.5
II	24	38.09	3	12.5	22	91.66	2	8.34
III	31	49.21	12	38.70	29	93.54	2	6.46

I & II t 30 d.f. = 3.857 $p < 0.001$ (Highly significant)

II & III t53 d.f. = 0.08 $p > 0.05$ (Not significant)

I & III t 37 d.f. = 5.6 $p < 0.001$ (Highly significant)

The ocular lesions prevalence was 91.66% and 93.54% respectively, in stage II & stage III. The prevalence of ocular lesions in stage I was only 37.5%. The mortality was highest in stage III patients which was 38.7%.

Duration of disease :-

Table VIII shows correlation of duration of illness to the outcome of disease. It is evident from the table that as duration of illness increased, prevalence of ocular involvement also increased.

TABLE VIII

PREVALENCE OF OCULAR LESIONS & DEATHS BY DURATION OF DISEASE

Duration of Disease	Total patients		Deaths		With ocular lesions		Without ocular lesions	
	No.	%	No.	%	No.	%	No.	%
0-1 month (Group I)	15	23.80	3	20	8	53.33	7	46.67
1-6 months (Group II)	37	58.73	11	29.72	35	94.59	2	5.41
7 6 months (Group III)	11	17.46	2	18.18	11	100	-	-

Group I & II t 50 d.f.=4.555 p $<$0.001 (Highly significant)

Group I& III t 24 d.f.=3.133 p $<$0.01 (Significant)

Group II & III t 46 d.f.=0.181 p $>$0.05 (Not significant)

It is evident from table VIII that ocular lesions prevalence was more in group II (duration $>$ 1 month but $<$ 6 month) and group III (duration of illness $>$6 month) It was 94.59% & 100% in group II & III respectively. The mortality was highest in group II which was 29.72%.

Table IX shows cases having history of preceding illness viz. measles, history of content of TB cases, lymph glands, invalvement, history of ear discharge, B.C.G. vaccination, raised E.S.R. & Lymphocytosis.

TABLE IX

INVOLVEMENT OF VARIOUS FACTORS IN TBM CASES

Factors	No.of patients	Percent
Measles preceding illness	4	6.34
History of contact of TB cases	11	17.46
Lymph glands involvement	13	20.63
History of ear discharge	6	9.52
B.C.G. vaccination history	2	3.17
Raised E.S.R.	19	30.15
Lymphocytosis	11	17.46

Table X shows the frequency of ocular affection in the present series.

TABLE X

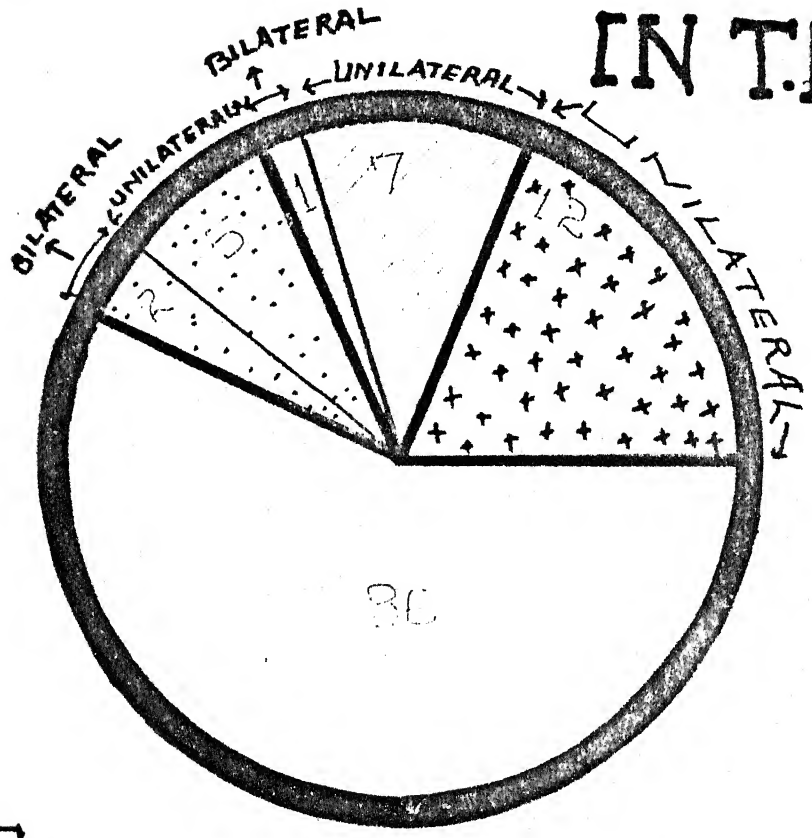
FREQUENCY OF OCULAR CHANGES IN TBM

Ocular changes	No.of cases	Percent
Optic nerve	51	80.95
Other cranial nerves	27	42.85
Pupillary changes	43	68.25
Anterior segment lesions	5	7.93

Table X gives us an idea about the frequency of the ocular manifestations. It is evident that optic nerve involvement was present in most of the cases (80.95%) cases where as anterior segment lesions were having low frequency (7.93%) cases.

Table XI shows frequency of other cranial nerves involvement in TBM patients of the present series (Fig. 3).

PI-DIAGRAM SHOWING CRANIAL NERVE PALSIES IN T.B.M.



NORMAL

FACIAL NERVE

OCULOMOTOR NERVE

ABDUCENT NERVE

Fig-3

TABLE XI

FREQUENCY OF VARIOUS CRANIAL NERVES IN TBM

Cranial nerve	Total cases		Unilateral involvement		Bilateral involvement	
	No.	%	No.	%	No.	%
Third (Ptosis only)	8	12.69	7	87.5	1	12.5
Sixth	7	11.11	5	71.42	2	28.58
Seventh	12	19.04	12	100	-	-

It is evident from table XI that 7th nerve palsy was the commonest of all (19.04%). Next in order of frequency was third nerve (12.69%). 7th nerve palsy was unilateral in all the cases where as third & sixth nerve palsies exhibited bilateral involvement too in 1 and 2 cases respectively. There was no case which showed multiple cranial nerve involvement.

The distribution of various pupillary abnormalities is shown in table XII.

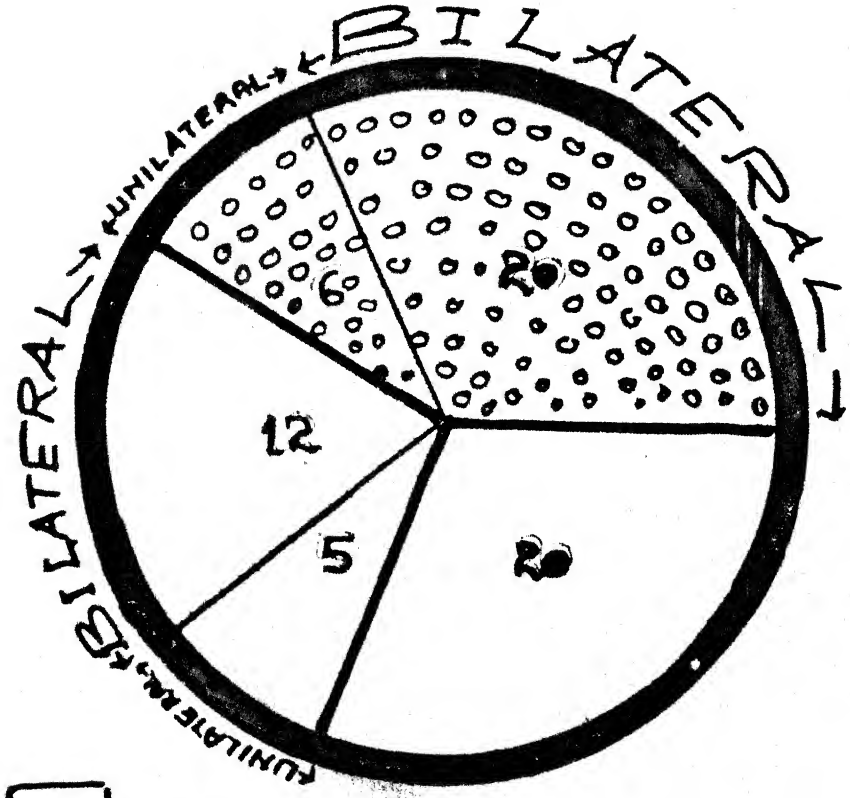
TABLE XII

DISTRIBUTION OF VARIOUS PUPILLARY ABNORMALITIES

Pupillary abnormality	Total patients		Unilateral		Bilateral	
	No.	%	No.	%	No.	%
Dilated & Fixed	26	41.26	6	23.07	20	76.93
Moderately dilated with sluggish reaction	17	26.98	5	29.42	12	70.58

It is evident from the table XII that dilated & fixed pupil seen in 41.26% out of which 20 cases had bilateral involvement. The moderately dilated

PI-DIAGRAM SHOWING PUPILLARY ABNORMALITIES IN T.B.M.



NORMAL

DILATED & FIXED

MODERATELY DILATED WITH SLUGGISH REACTION

Fig-4

pupil with sluggish reaction was seen in 26.98% cases in which 2 cases had bilateral involvement. (Fig.4).

Table XIII shows the distribution of fundus lesions in TBM patients (Fig.5).

TABLE XIII

DISTRIBUTION OF FUNDOSCOPIC LESIONS IN TBM PATIENTS

Fundoscopic appearence	Total cases		Unilateral		Bilateral	
	No.	%	No.	%	No.	%
Normal	12	19.05	-	-	-	-
Optic atrophy	20	31.74	6	30	14	70
Primary optic atrophy	4	6.34	-	-	4	100
Postneuritic atrophy	16	25.39	6	37.5	10	62.5
Papilloedema	16	25.39	3	18.75	13	81.25
Papillitis	12	19.05	4	33.33	8	66.66
Bitemporal pallor	2	3.17	-	-	2	100
Pale disc	1	1.58	1	100	-	-

It is evident from table XIII that maximum incidence of fundoscopic lesion was that of post neuritic atrophy & papilloedema each in 16(25.39%) cases. Next in order was papillitis in 12 (19.05%) cases. In cases of papilloedema, papillitis, primary optic atrophy, post neuritic atrophy and bitemporal pallor, majority of the cases showed bilateral involvement, whereas pale disc showed unilateral involvement.

The follow up study and management of various ocular problems was carried out in detail. The various results obtained are shown in table XIV, XV & XVI.

HISTOGRAM SHOWING FUNDUS LESIONS IN T.B.M

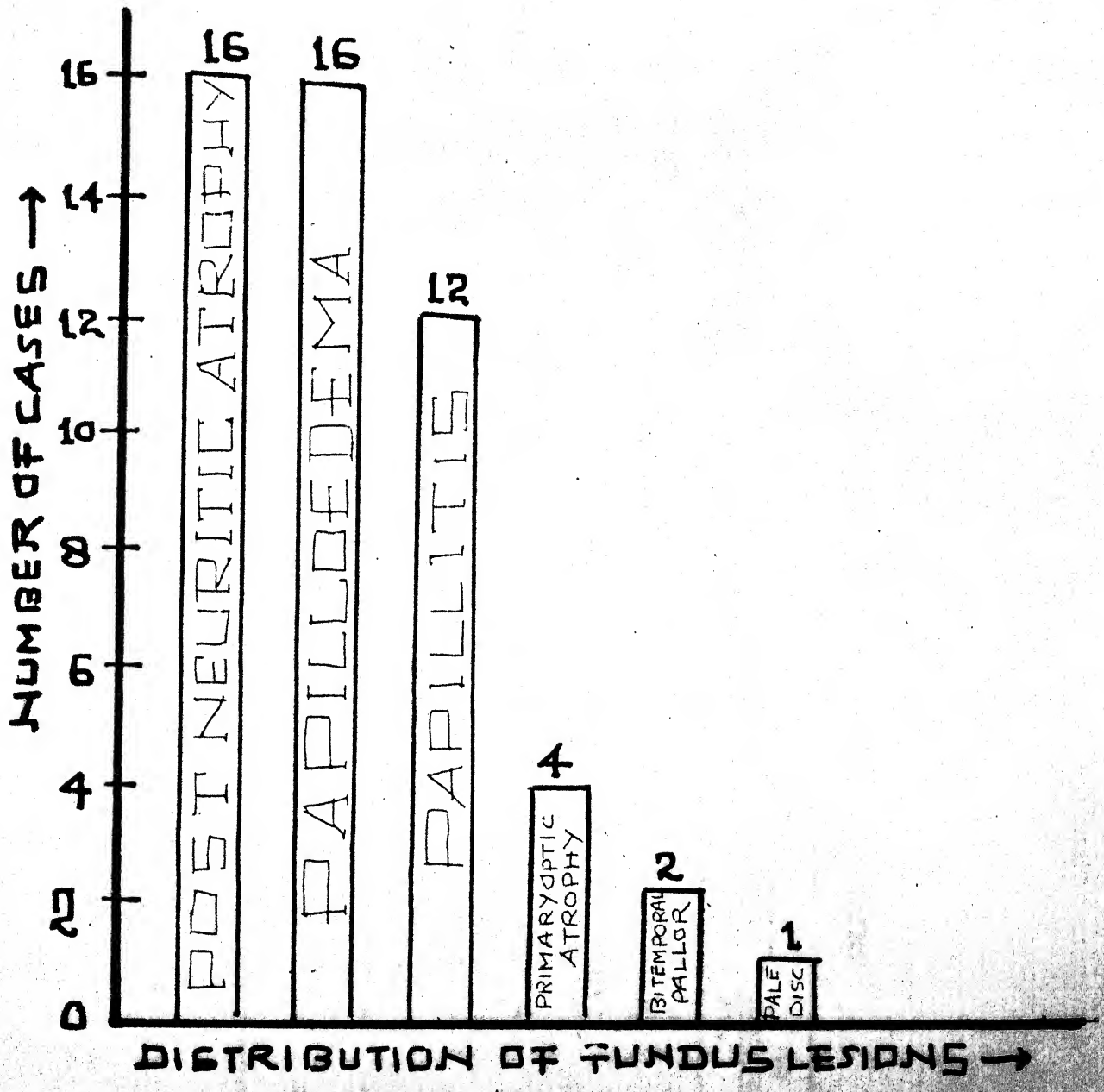


Fig-5

TABLE XIV

FOLLOW UP STUDY OF CRANIAL NERVES INVOLVEMENT

Cranial nerves	Total cases No.	%	Unilateral or Bilateral	Left against medical advice No.	%	Deaths No.	%	Recovery Complete No.	%	Recovery Partial No.	%	No Recovery No.	%
Third	8	12.69	Unilateral No.7(87.5%)	1	12.5	1	14.29	5	71.42	-	-	-	-
			Bilateral No. 1(12.5%)	-	-	-	-	-	-	1	14.29	-	-
Sixth	7	11.11	Unilateral No.5(71.42%)	-	-	2	28.57	3	42.85	-	-	-	-
			Bilateral No.2(28.58%)	-	-	-	-	-	-	2	28.57	-	-
Seventh	12	19.04	Unilateral No.12(100%)	2	16.66	3	30	6	60	1	10	-	-
			Bilateral Nil	-	-	-	-	-	-	-	-	-	-

TABLE XV

FOLLOW UP STUDY OF PUPILLARY ABNORMALITIES

Pupillary abnormality	Total patient		Left against medical advice		Death		Recovery		No recovery	
	No.	%	No.	%	No.	%	No.	%	No.	%
Dilated & fixed pupil	26	41.26	-	-	-	-	-	-	-	-
I.Unilateral	6	23.07	-	-	-	-	4	17.39	2	8.69
II.Bilateral	20	76.93	3	11.53	10	43.47	3	13.04	4	17.39
Moderately dilated with sluggish reaction	17	26.98								
I.Unilateral	5	29.42	-	-	-	-	4	26.66	1	6.66
II.Bilateral	12	70.58	2	11.76	2	13.33	5	33.33	3	20

TABLE XVI

PROGRESS & FOLLOW UP STUDY OF FUNDOSCOPIC & ANTERIOR SEGMENT LESIONS

Lesion	Total cases		Left against medical advice		Deaths		Recovered		Progressed to post neuritic atrophy				Remained same	
									Recovered		Not recovered			
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Conjunctivitis	3	4.76	-	-	-	-	3	100	-	-	-	-	-	-
Corneal Ulcer	2	3.17	-	-	-	-	2	100	-	-	-	-	-	-
Primary optic atrophy	4	6.34												
-Unilateral	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-Bilateral	4	100	1	25	-	-	1	33.33	-	-	-	-	2	66.66
Post neuritic atrophy	16	25.39												
-Unilateral	6	37.5	-	-	1	7.14	4	28.57	-	-	-	-	1	7.14
-Bilateral	10	62.5	2	12.5	4	28.57	2	14.28	-	-	-	-	2	14.20
Papilloedema	16	25.39												
-Unilateral	3	18.75	-	-	-	-	2	12.5	1	6.25	-	-	-	-
-Bilateral	13	81.25	-	-	8	50	3	18.75	-	-	2	12.5	-	-
Papillitis	12	19.05												
- Unilateral	4	33.33	-	-	-	-	3	27.27	-	-	1	9.09	-	-
- Bilateral	8	66.66	1	8.33	3	27.27	4	36.66	-	-	-	-	-	-
Bitemporal pallor	2	3.17	-	-	-	-	1	50	-	-	1	50	-	-
Pale disc	1	1.58												
-Unilateral	1	100	-	-	-	-	1	100	-	-	-	-	-	-
-Bilateral	-	-	-	-	-	-	-	-	-	-	-	-	-	-

It is evident from table XIV that facial nerve showed unilateral involvement only. Out of 12 cases, 2 cases left against medical advice, 3 cases (30%) died while 6 cases (60%) showed total recovery and 1(10%) case showed partial recovery after folloup. Next to it third nerve showed unilateral involvement in 7 cases & bilateral in 1 case only. Bilateral 3rd nerve palsy case recovered partially where as out of 6 unilateral involved cases 1 patient left against medical advice, 1 died & 5 recovered. In sixth nerve palsy there were 5 cases of unilateral involvement and 2 cases of bilateral one out of 2 bilateral 6th nerve palsy cases both of them recovered partially where as in 5 cases of unilateral involved cases complete recovery was in 3 cases & rest 2 died. Thus 6th nerve & 7th nerve, involvement showed high mortality. It is also observed that most of the cranial nerve patsies recovered in follow up study.

Table XV shows the details of various pupillary abnormalities in follow up study. It was observed that out of 26 total dilated & fixed pupil 6(23.07%) were having unilateral involvment where as 20(76.93%) were of bilateral ones. Out of six unilaterally dilated & fixed pupil, 4 cases recovered whereas 2 cases remained as such. In case of bilateral involvement, 3 cases left against medical advice, 3 recovered, 4 remained same and 10 cases died. Thus the mortality was highest in bilateral dilated & fixed cases that was 10 cases out of 17 cases of dilated & fixed pupil.

Out of 17 cases of moderately dilated with sluggish reaction 12(70.58%) cases were having bilateral involvement & 5 cases (29.42%) were having unilateral involvement. Out of 12 cases of bilateral involvement 2 left against medical advice, 5 recovered & 2 died. In unilateral involvement 4 cases recovered out of 5 cases.

The details observed in the follow up study of fundoscopic & anterior segment lesions is shown in Table XVI. The anterior segmentl lesions incidence was very low. They were conjuctivitis in 3 cases (4.76%) and corneal ulcer in 2 cases (3.17%). These all 5 cases recovered with treatment. Primary optic atrophy was in total 4 cases and all cases having bilateral involvement. Out of 4 cases 1 patient left against medical advice, 1 recovered & 2 cases remained same. In case of post neuritic atrophy out of 16 cases bilateral involvement was in 10 cases (62.5%). 6 cases of post neuritic atrophy recovered & total 5 cases died out of 16 cases. Thus it showed high mortality in comparison with primary optic atrophy. Papilloedema was observed in 16 cases. Out of total 16 cases, 13 cases were having bilateral oedema & 8 cases expired out of 8 survival cases 5 cases recovered, and 1 case progressed to optic atrophy which lateron recovered. Thus it was seen that cases having papilloedema showed grave progmosis & bilateral involvement in majority of the cases.

Papillitis was observed in 12 cases, among them 4 cases were having unilateral involvement & rest bilateral. Out of total 12 cases, 1 cases absconded, remaining 7 cases recovered & one case progressed to post neuritic atrophy which remained same. The mortality in papillitis was 27.27% (3 cases). One case of paledisc having unioacular involvement was noted which recovered. 2 cases of bitemporal pallor was present in the study 1 case recovered & other progressed to post neuritic atrophy which remained same even after treatment.

DISCUSSION

D I S C U S S I O N

The present endeavour has been based on, a study of ocular problems and their management in paediatric patients of Bundelkhand Region including 63 patients of TBM. The study was conducted at M.L.B.Medical College, Jhansi between July 1983 to May, 1984.

The primary aim of our study was to find out the prevalence of ocular lesions of tuberculous meningitis in paediatric patients of Bundelkhand Region, to decide the prognosis of disease and to prevent the dreading sequelae of blindness by early detection of complications and their management . With these objectives in view, a detailed ophthalmoscopic study was carried out and various associated factors influencing prognosis of the disease were studied in detail in each case. Management and follow up study was carried in each case having ocular lesion. Statistical analysis was done where ever needed.

Based on observations depicted in table I to XVI, various inferences have been drawn and discussed under different headings.

Prevalence of ocular lesions :

The majority of our cases of TB belonged to group 0-4 years of age (preschool children). The incidence in this age group was 80.95%. There was preponderance of male cases over female cases in this study. Out of total 63 cases the female cases in this study were only 12(19.04%)

in number. Rao (1972) described that in patient statistics from children's medical wards of hospitals in different parts of India reveal that TBM cases from 1-4% of the total pediatric medical admission and majority of cases are encountered in the preschool age group because of exposure to infection in infants and young children in developing countries. The preponderance of male cases over female cases in our study is suggestive that parents are more health conscious for the male children in our community, they are readily taken to the hospital for consultation resulting in an apparently higher incidence in males.

The ocular prevalence in our study observed was 85.71%. Similar results were in the past study viz. Isente Ivan (1950) reported 95% ocular involvement and Mooney (1956) 72% ocular sequelae. In recent studies too, higher incidence of ocular lesions has been reported. Gupta (1976) reported 80% optic nerve involvement in paediatric patients of TBM in Jhansi district. Verma et al (1981) observed 70% ocular prevalence. The reason for higher ocular prevalence is possible due to overcrowded living condition, low socio-economic status, malnutrition, ignorance, illiteracy, poverty and failure to get prompt and adequate therapy in the patients of Bundelkhand Region.

Clinical picture of the disease :-

The important signs & symptoms observed in this study were fever 85.71%, vomiting 58.73%, altered sensorium 82.53% and meningeal signs 76.19%. Nelson (1975)

in his text book of pediatrics have described the symptomatology in similar way, the important clinical features being fever, vomiting, altered sensorium, convulsions, meningeal signs, motor deficit, various cranial nerve paralysis and fundal changes.

Relationship of various ocular lesions and mortality to factors influencing the prognosis of disease.

Socioeconomic status - Majority of cases of TB were from lower socio-economic class (Srivastava et al 1981 classification). The patients in class III, IV & V were 28.57%, 36.50% and 23.81% respectively. The ocular involvement in class III & IV was 83.33% & 93.30% respectively whereas in class V all cases were showing ocular involvement.

Nutritional status - Most of the cases in this study were having poor nutritional status (84.13%). In poor nutritional group ocular prevalence was 94.33% in comparison to fair nutritional status group i.e. 40% only, it is much higher. The mortality in poor nutritional status group was 25.39% where as in other group, there was no death. Statistical analysis revealed high significance between the two groups (t 61 d.f. = 5.4; $p < 0.001$).

Stage of disease - The cases in this study were mainly in stage II & III of the disease. Only 8 cases (12.69%) were in stage I. The ocular prevalence in stage II & III was

91.66% and 93.34% respectively whereas it was only 37.51% in stage I. The mortality was highest among stage III patienhs. The statistical analysis revealed high significance between ocular changes in stage I & II and stage I & III (Stage I & II t 30 d.f.=3.857; p $<$ 0.001 /Stage I & III t 37 d.f.=5.6 p $<$ 0.001).

Duration of illness-duration of illness was divided in 3 groups in this study Group I (duration of illness $<$ 1 month), Group II (duration of illness $\geq$ 1 month $<$ 6 months), and Group III (duration of illness $\geq$ 6 months). It was noted that ocular prevalence in group I & II was 53.33% and 94.59% where as in group III all% the cases were having ocular involvement. This inferred that neurological damage is directly proportional to the duration of illness. Statistical analysis revealed significance between group I & II and group I & III (Group I & II t 50 d.f. = 4.55; p $<$ 0.001/ Group I & III t 24 d.f.=3.133; p $<$ 0.01). Dikshit & Singh (1976) reported similar inferences regarding various factors influencing prognosis of tuberculous meningitis. It was observed in their study that mortality & morbidity was highest if the child was less than 3 years of age, in III stage of disease (MRC classification), malnourished, belonged to low socio-economic group, ill for more than 9 month. In their study poor nutritional cases were having 59% neurological damage in comparison to 29% only in fair nutritional group.

There was 41.7%, 35% and 37.5% morbidity in low socio-economic groups of III, IV & V respectively. When duration of illness was less, the damage was 35.70%, while it increased to 67.50% when it was more than 1 month.

Ocular problems :-

Cranial nerve palsies- In this series cranial nerve involvement was observed in 42.85% cases. The various cranial nerves involved were VII (Facial nerve), III (oculomotor nerve) and VI (abducent nerve) in order of frequency. The incidence of these nerves was facial nerve in 19.04% cases, oculomotor nerve in 12.69% cases and abducent nerve in 11.11% cases. Out of 8 cases of oculo-motor nerve 7 cases showed unilateral involvement whereas one case showed bilateral involvement. Similarly out of 7 cases of abducent nerve 5 cases having unilateral involvement & 2 cases bilateral. In contrast to oculomotor and abducent nerves, facial nerve involvement was seen unilaterally in all the cases. Saxena and Tomar (1970) reported similar findings. The incidence of cranial nerve palsies in their series was 47%. Out of these 47%, facial nerves palsy was seen in 23.5% cases where as oculomotor and abducent nerves were involved in 11.7% cases each. Thaper et al (1968) have reported low incidence of cranial nerve palsies i.e. 17% only (8% facial, 5% abducent and 4% oculomotor).

Bhatnagar & Srivastava (1961) suggested that the paralysis of ocular muscles results from infiltration of III and VI nerves at the base of brain. However bilateral

VI nerve palsies may result from raised intracranial tension.

Pupillary abnormalities :-

The prevalence of pupillary abnormalities in this series was 68.25%. Out of these total pupillary abnormalities dilated & fixed pupils were noted in 41.26% cases. Among them 20 cases were having bilateral involvement where as unilateral involvement was noted only in 6 cases. Moderately dilated with sluggish reactions of pupil was observed in 26.98% cases. Out of total 17 such cases, 12 cases showed bilateral involvement. Similarly Desai & Ankelsaria (1967) reported the various pupillary abnormalities in 63.82% cases. Khatua (1961) reported a much higher incidence of pupillary abnormalities in TBM cases that was 76% whereas Misra & Gupta (1962) reported pupillary abnormalities in 25% cases only.

Walsh (1957) suggested the dilated & fixed pupils were seen in those cases in which optic atrophy had been set up. These pupils may also be due to cerebral irritation by raised intracranial tension.

Anterior segment lesions :-

The ocular prevalence for anterior segment legions in this series was very low only 4.76% cases had conjunctivitis and 3.17 had corneal ulcer. Verma et al(1981) reported similar results. They reported 6% cases of corneal ulcer and conjuctivitis each. Many other workers in the field have however reported no anterior segment lesions. It is postulated that conjuctivitis may be due to allergy or infection and corneal ulcer may result from exposure

keratitis. These lesions may be due to poor body resistance and neglected eye care.

Fundoscopic lesions :-

The ocular prevalence of various fundoscopic lesions viz. papilloedema papillitis, optic atrophy, bitemporal pallor and pale disc, was 80.95% in this series Desai & Ankelsaria reported similar fundus changes that was in 74.46% cases. Bhatnagar and Srivastava (1961) reported 43.3% optic nerve involvement only.

Papillitis and papilloedema :-

In this series papillitis was observed in 19.05% cases. Out of total 12 cases of papillitis, 4 case showed unilateral involvement where as 8 cases showed bilateral involvement. Papilloedema showed higher incidence in comparision to papillitis. It was observed in 25.39% cases. Out of total 16 cases of papilloedema 13 cases showed bilateral involve,ent. Mooney (1956) reported papilloedema in 26% cases and retrobulbar neuritis in 3.1% cases only. Bhatnagar & Srivastava (1961), reported a higher incidence of papilloedema & papillis viz 16.6% & 13.3% respectively. Similarly Misra & Gupta (1962) reported higher incidence of papillitis (35%) in comparison to papilloedema (7.5%). Mooney (1956) suggested that the pathology in reversible papilloedema was external hydrocephalus where as in progressive papilloedema the cause is tuberculous arachnoiditis which in turn interferes with the circulation of C.S.F.The

pathologic process of papillitis is supposed to be optico-chiasmatic arachnoiditis.

Optic atrophy :-

The prevalence of optic atrophy in this series was 31.74% Saxena & Tomar (1970) reported optic atrophy in 29.1% cases similarly. The optic atrophy observed in this series was grouped in 2 forms.

I- Primary optic atrophy :-

The primary optic atrophy was seen only in 6.34% cases. All the cases were having bilateral involvement. Saxena & Tomar (1970) reported 12.7% primary optic atrophy Cases Lothe et al (1972) reported a higher incidence of primary optic atrophy that was 56% . Verma et al (1981) stated that optic atrophy may be due to internal hydrocephalus of third ventricle causing pressure on optic Chiasma.

II- Post neuritic optic atrophy - It was observed in 25.39% cases out of 16 cases 10 cases were having bilateral involvement. Verma et al (1981) reported post neuritic atrophy in 12% cases.

Post neuritic atrophy observed in this series was supposed mainly due to preceding papilloedema or papillitis.

Bitemporal pallor :- It was reported in 3.17% cases Desai & Ankelsaria (1967) reported bitemporal pallor in 4.2% cases and suggested that it may be due to compression type of lesion at the level of chiasma. Verma et al (1981)reported bitemporal pallor in 10% cases.

Paledisc - It was seen only in 1 case (1.58%) having unilateral involvement Desai & Ankelsaria (1967) suggested that the pale disc is suggestive of reduction in blood supply to optic nerve. Only Desai & Ankelsaria reported Paledisc (12.76%) lesion in the field of ocular manifestations of THM.

Management & Follow up study of ocular lesions:-

Cranial nerve involvement - Out of total 12 cases in which facial nerve was involved, 2 cases left against medical advice. In remaining 10 cases 30% expired 60% cases recovered totally and 10% partially.

In abducent palsy out of 7 cases 28.57% expired, 42.65% having unilateral involvement recovered totally where as 28.57% cases - having bilateral involvment recovered partially. In 8 cases of oculomotor nerve palsy, 1 case left against medical advise. Out of remaining 7 cases 14.29% expired and 71.42% cases - having unilateral nerve palsy recovered completely where as 14.29% cases- having bilateral involvement recovered partially.

Thus it is evident that in cranial nerve palsies recovery was almost complete. The mortality was high in facial nerve and abducent nerve palsy showing grave prognosis. Bhatnagar & Srivastava (1962) reported complete recovery in oculomotor nerve involvement where as 50% mortality was seen in abducent nerve involvement . Desai & Ankelsaria (1967) reported complete recovery in facial

nerve palsy. Where as in oculomotor nerve palsy all cases having bilateral involvement 50% recovered partially, & 50% completely. In case of Sixth nerve, 16.66% cases recovered partially and 83.34% cases recovered completely. Verma et al (1981) reported that out of total oculomotor nerve palsy 16.6% expired where as in abducent nerve palsy mortality was 33.33%.

Pupillary abnormalities -

Out of 26 cases of dilated & fixed pupil 43.47% expired, 30.43% recovered and 26.08% cases remained same. The cases which remained same gives an idea that in these cases optic atrophy was set up which did not recover even after treatment. The mortality was high in dilated & fixed pupil cases. Verma et al (1981) also reported 25% mortality in dilated & fixed pupil.

Among 17 cases of moderately dilated pupil with sluggish reaction 13.33% expired, 60% recovered and 26.66% showed no recovery. Bhatnagar & Srivastava (1961) reported complete recovery in moderately dialted pupil with sluggish reaction. In our cases the cases which did not recover means that these cases were not relieved from optic nerve lesions even after treatment.

Anterior segment lesion - All the cases of conjunctivitis and corneal ulcer recovered after treatment. It infers than proper eye care is necessary during the course of disease to avoid anterior segment lesions.

Fundoscopic lesions -

Papilloedema & papillitis - Out of total 16 cases of papilloedema 50% cases expired which were having bilateral involvement, thus showing grave prognosis 31.25% cases recovered while 18.75% cases progressed to postnueritic atrophy. Among the 3 cases which progressed to post neuritic atrophy, 1 case recovered and 2 cases remained same. In 12 cases of papillitis 1 case left against medical advice and 27.27% cases expired, 63.63% cases recovered and 9.09% cases progressed to optic atrophy which did not recover in follow up stidy. Thus it is evident that papillitis cases were having better prognosis in comparison to papilloedema. Desai & Ankelsaria (1967) reported that 50% cases of papilloedema recovered. 25% expired and 25% progressed to optic atrophy where as in papillitis 69.2% cases recovered, 23% cases expired and 7.69% cases progressed to post neuritic atrophy. Thus our findings very much simulates to Desai & Ankelsaria series. Verma et al (1981) reported 75% mortality in papilloedema and 33.33% in papillitis. Thus papilloedema is having grave prognosis. Post neuritic optic atrophy- Out of 20 cases of post neuritic optic atrophy, 2 cases left against medical advice, 35.71% expired, 42.85% recovered and 21.42% remained same.

Primary optic atrophy - Out of 4 cases of primary optic atrophy 1 case left against medical advice, 33.33% recovered and 66.66% remained same.

Bitemporal pallor & paledisc-50% cases of bitemporal pallor and total 1 case of pale disc recovered after judicious therapy.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study was undertaken to evaluate the ocular lesions in tuberculous meningitis of paediatric patients in Bundelkhand region, to decide the prognosis of disease and to prevent the dreading sequelae of blindness by early detection of complications and their management. The study was carried out in the Department of Ophthalmology in active collaboration with the department of Paediatrics, M.L.B. Medical College, Jhansi, over a period of 11 months from July 1983 to May, 1984. 63 children upto the age of 13 years, suffering from tuberculous meningitis (TBM) were selected for this study. These children admitted to the paediatric ward of M.L.B. Medical College, Jhansi belonged to Jhansi district and nearby districts of Uttar Pradesh and Madhya Pradesh forming the Bundelkhand Region. A detailed clinical examination was carried out. Due importance was given to obtain the history of contact, to record B.C.G. vaccination and to evaluate nutritional status by dietary history and weight of the child. The socioeconomic status of the patient and history of preceding illness was also noted in each case. A detailed ocular examination was carried out in each case with the help of Binocular loupe, bright torch light and Keeler's Direct ophthalmoscope. The various ocular problems were managed & followed up during hospital stay and after discharge from the hospital.

Ocular manifestations observed in cases of TBM were mainly in the form of cranial nerve palsies, various pupillary abnormalities and foundoscopic lesions. The various foundoscopic lesions were papilloedema, papillitis, opticatrophy, bitemporal pallor and pale disc. Anterior segment lesions in the form of conjunctivitis and corneal ulcer were noted in very few cases.

Most of the patients having ocular problems were relieved with cortisones therapy (given by oral, intramuscular intravenous and retrobulbar routes depending on the condition) vasodilators, hyperosmolar agents and neurotonics, along with antitubercular treatment as adjuvant to the therapy.

In the light of present work following points summarizes the salient features of our study.

1. Majority of cases of TBM in our study belonged to pre-school children group. The prevalence rate was 80.95% in 0-4 years age group.
2. There was preponderance of male cases over female ones.
3. Out of 63 cases ocular involvement was seen in 54 cases (85.71%) while 9 cases showed no ocular problem.
4. The mortality in this series was 25.39%. It was interesting to note that all the cases which expired were having ocular problems.
5. It was observed that mortality and morbidity was highest if the child was -malnourished, belonged to low socio-economic status, in stage III & IV of disease and ill for more than 1 months.

6. Anterior segment lesions were seen only in 7.93% cases. All cases recovered after giving treatment.

7. Cranial nerve involvement was seen in 42.85% cases. The distribution of various cranial nerve involvement and follow up results were as.

(I) Facial - It was involved in 19.4% cases all cases were unilaterally involved. Majority of cases got relieved Mortality was 30%.

(II) Oculomotor nerve - Incidence was 12.69%. The involvment was mostly unilateral. Mortality was 14.29%, while majority of cases recovered.

(III) Abducent - It was involved in 11.11% cases. Majority of cases had unilateral involvement and showed marke-d recovery. Mortality was 28.57%.

8. Pupillary abnormalities - The prevalence was 63.82%. Pupillary abnormality was observed in two forms -

(I) Dilated & Fixed - It was seen in 41.26% cases. Majority of the cases had bilateral involvement. Mortality seen in this abnormality was 43.47%. Out of total followed cases, 30.43% recovered & 26.08% remained same.

(II) Moderately dilated with sluggish reaction was observed in 26.98% cases. Out of total followed up cases 13.33% expired 60% recovered and 26.66 remained same. In this group also there was bilateral involvement in most of the cases.

9. Fundoscopic lesions - These were seen in 80.95% cases. The various lesions & their follow up results were as-

(I) Papilloedema - It was seen in 25.39% cases majority of cases showed bilateral involvement. Out of total followed up cases 50% expired, 31.25% recovered and 18.75% cases progressed to optic atrophy. Out of the cases which progressed to optic atrophy, 12.5% did not recover whereas 6.25% cases recovered.

(II) Postneuritic atrophy - It was seen in 25.39% cases mostly bilaterally. Out of total followed up cases, 35.71% expired, 42.85% recovered and 21.42% remained same.

(III) Primary optic atrophy - It was observed in 6.34% cases, out of total followed up cases 66.66% remained same and 33.33% improved.

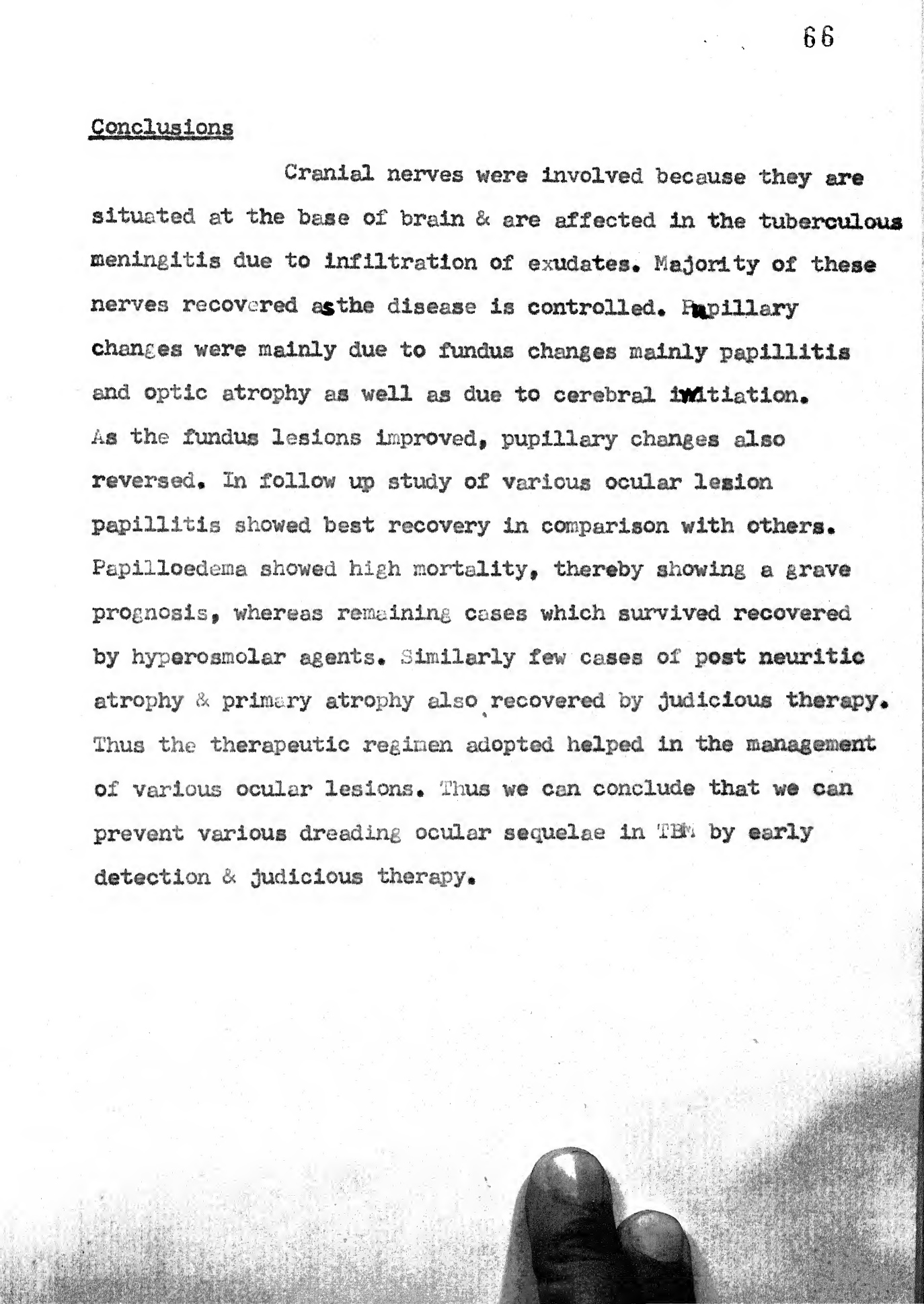
(IV) Papillitis - It was observed in 19.05% cases mostly bilaterally. Among total followed cases 27.27% expired, 63.63% recovered 9.09% cases progressed to optic atrophy.

(V) Bitemporal pallor - It was seen only in 3.17% cases. Out of 2 cases 1 progressed to optic atrophy & one recovered.

(VI) Pale disc - It was seen only in one case (1.58%) which lateron recovered.

Conclusions

Cranial nerves were involved because they are situated at the base of brain & are affected in the tuberculous meningitis due to infiltration of exudates. Majority of these nerves recovered asthe disease is controlled. Papillary changes were mainly due to fundus changes mainly papillitis and optic atrophy as well as due to cerebral irritiation. As the fundus lesions improved, pupillary changes also reversed. In follow up study of various ocular lesion papillitis showed best recovery in comparison with others. Papilloedema showed high mortality, thereby showing a grave prognosis, whereas remaining cases which survived recovered by hyperosmolar agents. Similarly few cases of post neuritic atrophy & primary atrophy also recovered by judicious therapy. Thus the therapeutic regimen adopted helped in the management of various ocular lesions. Thus we can conclude that we can prevent various dreading ocular sequelae in TBM by early detection & judicious therapy.



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APPENDIX

APPENDIX -I

WORKING PROFORMA FOR EXAMINATION

Topic - Ocular problems and their management in paediatric patients of Tuberculous Meningitis in Bundelkhand Region.

Chief Guide - Dr. G. D. GUPTA, M.S.,D.O.M.S.
Co-Guide - Dr. R. S. SETHI, M.D.,D.C.H.
Worker - AJAY KUMAR SAXENA

M.R.D.No.:

Name : Sex : Male/Female

Age :

Father's Name :

Address :

Occupation : Father Mother

Total Income of family :

Birth order of child :

Geniological Tree :

Dietary History:

	Started at age	Upto age
Breast Milk		
Artificial Milk only		
Added artificial Milk		
Solids added		

Present nutritional status :

Immunisation History:

Small pox	B.C.G.	Polio (oral)	DPT (Triple)

Antenatal and natal history :

Full term / premature

Normal / Forceps / Caesarean

Postnatal history (First 4 weeks) :

No problem / Fever / Sepsis / Jaundice / Cyanosis.

Mile Stones:

	Age
Social smile	
Neck holding	
Sitting	
Crawling	
Standing	
Walking	
Speaking	
Monosyllables	
Hadling of spoon	

Family history :

Present illness :

Fever

Vomiting

Headache

Change in disposition

Convulsions

Alteration in consciousness

Others

Past illness :

Definite H/O Primary complex

H/O Pertussis

Measles

Ear discharge

Head injury

Convulsions

Worm infestations

Others

History of contact of open tuberculous cases :

General examination :

General appearence - Healthy / Malnourished

Pulse

Temperature

Lymphadenopathy - Significant / Insignificant

Weight

Others

Systemic examination :

1. C.N.S.

Level of consciousness :

Conscious

Stage I - Stupor

Stage II- Lightcoma

Stage III-Deepcoma

Stage IV -Patient flacid and apneic

Posture
(Normal, Decorticate, Decerebrate).

Speech

Gait

Signs of Meningeal Irritation

Neck rigidity

Kernigs sign

Brudzinskɸi's sign

Cranial Nerves (Affected / not affected)

II (Optic nerve)

III (Oculomotor nerve)

IV (Trochlear nerve)

VI (Abducent nerve)

VII (Facial nerve)

Others

Motor System	Lower limb		Upper limb	
	Right	Left	Right	Left
(I) Power				
(II) Bulk				
(III) Tone				
(IV) Co-ordination				
(V) Involuntary movements				

Sensory System :

Reflexes	Right	Left
I. Superficial		
a. Corneal		
b. Conjunctival		
c. Pupillary (Direct & Consensual)		
d. Abdominal		
e. Cremasteric		
f. Planters		
II. Deep		

Cerebellar Signs

Extrapyramidal signs

2. C.V.S.

3. Respiratory

4. Abdominal

Investigations :

I.C.S.F.

Tension

Colour

Coagulum

Protein

Sugar

Chloride

Cells

Others

II. Blood

TLC

DLC P L E M

ESR

Hb

III. Urine

Albumin

Sugar

IV. X-ray

Chest

Skull

V. Others

Ocular complaints :

1. Diminished vision

Onset: Sudden/Gradual

Duration:

2. Pain
3. Redness
4. Watering
5. Irritation
6. F.B.Sensation
7. Discharge
8. Others

OCULAR EXAMINATION:

(I) Ocular

		Right	Left
I.	Face		
II.	Eyebrows and eye lashes		
III.	Eye lids		
IV.	Ptosis		
V.	Nystagmus		
VI.	Squint		
VII.	Laerimalsac		
VIII.	Conjunctiva		
IX.	Cornea		
X.	Pupil		
XI.	Anterior chamber		
XII.	Iris		

	Right	Left
XIV. Ocular movements		
XV. Tension		
XVI. Vision		

(II) Ophthalmoscopy

	Right	Left
1. Media		
2. Opticdisc		
a. Size		
b. Shape		
c. Colour		
d. Margins		
e. Cup		
f. Others		
3. Other details		
I. Retinal Vessels		
II. Macula		
III. Retina		
IV. Choroid(Tubercles)		
V. Others		

Ophthalmoscopic diagnosis :

Management :

I. Antitubercular treatment

II.Others

Follow up fundoscopy :